













Ontario

Royal Commission on Matters of Health  
and Safety Arising from the Use of  
Asbestos in Ontario

Submissions

v. 7



## INDEX OF WRITTEN SUBMISSIONS

BINDER VOLUME NUMBER: 7

Brief No.	Author	Author Category	Subject Matters	I or Phase III Hearing Requested	Legal Stand Reque
63	International Assn of Heat & Frost Insulators & Asbestos Workers	Labour	II Workplace VII Workers' Compensation	Yes June 8/81	No
64	Mrs. Betty Glaser	Labour	II Workplace VII Workers' Compensation	Yes June 8/81	Yes via AVC
65	Bendix Corporation	Industry	II Workplace VII Workers' Compensation	No	No
66	Hamilton Labour Council	Labour	II Workplace VII Workers' Compensation	No	No
67	Alberta Lever	Labour	II Workplace	No	No
68	Wolfgang Palleske	Labour	VII Workers' Compensation	No	No
69	Workmen's Compensation Board, Ontario	Government	VII Workers' Compensation	No	No
70	G.W. Cousineau	Labour	VII Workers' Compensation	No	No
71	Floyd Lefebvre	Labour	VII Workers' Compensation	No	No













## Insulators Health Hazard Program


511 Machinists Building  
1300 Connecticut Ave., N.W.  
Washington, D.C. 20036  
(202) 785-2150

As you know, the Asbestos Workers have always been deeply concerned, highly interested and vitally aware of matters and materials in reference to occupational safety and health. We are constantly active in instructing our local unions, officers and members in all possible facets of job safety and health, not only as how it may affect our members but also how it may affect workers in close proximity when we are working with carcinogenetic or toxic materials or products.

It has been a strict standard policy of our organization for many years to fully investigate the true cause of death of every member. This action necessitates (in a large percentage of the cases) changing the cause of death legally as stated on the death certificate. This further qualifies many more widows and dependents for Workers Compensation or other benefits for job-incurred injuries or occupational disease. This is investigated by Mt. Sinai School of Environmental Medicine under the direction of Dr. Irving J. Selikoff.

In addition to the above, many local unions and individual members who are experiencing job-related health problems receive authorizations at their request from this office for their signature. In turn the authorizations are forwarded to Dr. Irving J. Selikoff who consequently obtains from their physicians the necessary x-rays, medical histories and other needed medical data which assists him to diagnosis whether or not the member is disabled or suffering from an occupational disease or injury. This office then advises him of the proper procedures to follow in regards to financial relief, Workmans Compensation benefits, etc.

Regarding contract or working agreement language, we stress very forcefully that the employer remain liable for all safety and health issues



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with provincial, federal and state regulations being followed. At this office we strive to review all proposed contract language. This prevents any liability against the Local and International Unions.

I strongly believe all fatalities in all Local Unions should be thoroughly investigated to determine the true cause of death by expert medical professionals. Too often we find a widow does not request an autopsy or ill-advised medical personnel perform the wrong type of post mortem. This results in reducing or making it impossible for the widow to collect the benefits she so justly deserves.

Statistics clearly indicate that approximately 50% of all deaths in our organization are caused by asbestos-related disease; 20% from lung cancer, approximately 12% from mesothelioma (peritoneal and plueral, with about twice as many peritoneal as plueral cases); and the remaining causes being cancer of the G.I. tract, larnyx, kidney, cancer all sites and 8% asbestosis. Although it is extremely difficult to establish the extent of the average age when the worker enters the industry, we estimate our members have a 15-year shorter life span. Based on all statistics to-date and at the current earning power of our members, we estimate the average loss of income due to early total disability and death from asbestos-related disease to be between \$350,000 to \$400,000.

Planning is now being finalized for an Early Cancer Detection Program. This will include thorough revised physical exams for all our members in the United States and Canada who have had over 30 years experience in our industry plus all members who are currently experiencing health problems. This will involve about 4-5,000 members.





New examination techniques including additional blood tests involving the white blood corpuscles, D.N.A. and the thymus gland output will be added to existing examination procedures. Also to be included will be possible treatment with current and new experimental drugs and other new procedures. The blood samples taken will be flown daily to Mt. Sinai in New York City for analyzation by hemotologists of the School of Medicine.

We will have a total of about 25 examination sites in the United States and Canada. Transportation and expenses to the sites will be provided by the Insulators Health Hazard Program. We firmly believe this will be the largest exam program of this type ever attempted. Of course, the results will be available to the whole society.

In addition, one of the larger pharmaceutical companies has volunteered to donate a supply of Interferon and Thymasin. This is welcomed wholeheartedly as we expect to find a considerable amount of cancer and hopefully it can be treated.

It is probably common knowledge that our members' exposure to asbestos has been greatly reduced. However, the Asbestos Workers are becoming deeply involved in asbestos removal, maintenance and repair of existing facilities. Also between 66% and 75% of asbestos products being used today are in construction. This calls for continual expanding educational programs in the proper use of protective clothing and respiratory equipment.

As a member of the O.S.H.A. Advisory Committee for Construction, the other labor members and I have continually stressed the need for a lower exposure level -- 1 fib. per CC for asbestos exposure. However, as most everyone knows, the cutbacks on O.S.H.A. regulations, standards, enforcements and job safety and health investigations are only now starting to immerge.





I have been informed three times that our initial meeting under the new administration has been postponed. This comes as no surprise to me as the current and new Under-Secretary for O.S.H.A. has, as head of his construction company, received 6 serious citations and 42 other non-serious citations. I believe this in itself speaks of what we have already seen or heard of the restrictions we can expect from this government agency. It is my opinion that in order to continue to protect all the working people in the United States under the current administration, we must resort to and use every tactic that organized labor has used so successfully in the past.

I firmly believe that any product or material that is made or used in this country or abroad should be of high quality and of danger to no one. For too long we have endured situations where our own bodies have or will become toxic waste receptacles.

The popular slogan of today is de-regulate industry. In all good conscience, who can say that too many worker protection controls have been imposed on industry.

I thank you.

Roy J. Steinfurth  
Administrator  
Insulators Health Hazard Program  
International Association of Heat & Frost  
Insulators and Asbestos Workers





Penwood Hall, Bldg.  
Scarborough, Ontario

May 26, 1981

WC  
64

Gentlemen of the Royal Commission:

I am here on behalf of my husband Gus Glaser. I was here in March and described my husband's condition. Gus has asbestosis.

Gus was re-examined in March by W.C.B. To this date we have not heard from them.

On April 21, we had a Hearing before the Appeal Board. My husband was represented by Mr. Daniel Ublansky legal council for the Energy and Chemical Workers Union. We were told by the Appeal Board we would hear from them sometime in July. Why must so much time elapses before we hear from them. By the time they get around to my husband's case, he will only be a number.

Mr. Ublansky had received Gus's records from W.C.B. to see how they rated his lung condition. Apparently W.C.B. do the ratings by numbers -2-3 ect. Now what we found strange is that Gus's condition through the years had no pattern. His ratings varied. It seemed to us if they were pulled out of a hat. You do not start out with a high number, then a low one. You do not get better with asbestos and this is what appeared to be happening to Gus on paper. This does not appear to me to be quite proper.

Briefly I would like to submit these two cases, as an indication of how well W.C.B. does their job. — *these refer to page 1 & 2 - separate*

quote from Professor (name withheld) Dept. of Preventive Medicine, University of Toronto. It is well known that exposure to high levels of asbestos for an adequate period of time may cause a variety of chest deceases including cancer of the lung. It is also known that continued fibrosis of the lungs, as can occur from asbestos, can affect the heart and may cause it to fail.

Example - MIKE LITKE

Dr. W.E. Cooke, an English Physician, noticed as far back as early as 1900, a 33 year old man who worked for 14 years in one of the earliest asbestos-textile factories, died, the cause of death, scarring and thickening (fibrosis) of the lung tissues in which asbestos fibres were embedded. His description of the extensive lung scarring and loose strands of abnormal fibrosis tissue connecting the lungs to the plueral membrane surrounding them also referred to solid yellowish brown "curious bodies" in the scarred area. Of these he said we have never seen anything parallel to this in pneumoconioses due to other dusts. We cannot think there is any reasonable doubt that the particles in the lung are the heavy, brittle, iron-containing fragments of asbestos fibre.

Antibiotics were discovered and reduced the death rate from pneumonia and other infectious pulmonary diseases to which asbestosis sufferers were particularly susceptible.





## Case 1

CLAIM - S 10186035  
MICHAEL LITKE - 23 Harwood Ave. N.  
R.R.#1, Pickering  
Ontario

First sign of Fibrosis - 1972 - Recognized 1974

Mike received letter from W.C.  
July 9, 1976 - Enclosed -(Copy) No change -  
July 27, 1976 - Mike entered hospital with an acute chest condition.  
Was diagnosed as a heart attack.  
Enclosed - copy- *M/Litke*  
Aug. 16, 1976 - Xray, showed tumor on lung.  
Doctors operated in August 1976.  
Was magligment - asbestosis.

Mike died, the following June 1977. Full of cancer, 100%. Autopsy was done.

Mike died within a year after he was operated.

## Case 2

ALFRED GLASER

In the fall of 1979, he complained of severe abdominal pain. Went to several doctors, was Xrayed and told he had gall bladder trouble. Was treated for gallbladder several weeks, until the pain was so severe, the doctors where not sure what was wrong.

He was in Ajax hospital for several weeks, when one doctor suspected cancer and suggested that perhaps Alfred should go to Toronto General Hospital and have an exploratory.

He was operated on and found to have Abdominal Cancer. Cancer in the colon and in the intestines. Nothing could be done. Cause - asbestosis. He went from 225 lbs. to a mere 135 lbs.

## NOTE

Alf died in August 1980.  
Alfred was Xrayed yearly and was told nothing was wrong with him and yet he lived only 9 months after the exploratory operation.



2 Bloor Street East  
Toronto, Ontario  
M4W 3C5  
Telephone (416) 965-8804

The Workmen's Compensation Board



July 9, 1976.

Mr. Michael Litke,  
23 Harwood Avenue, N.,  
R. R. #1,  
PICKERING, Ontario.

Claim: S10186035

*Lung Tumor shown*  
*Aug 16*  
*XRAY*

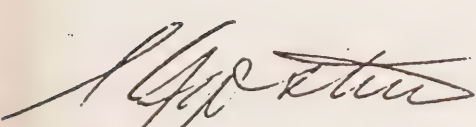
The Advisory Committee on Occupational Chest Diseases reports there is no change in your classification as a silicotic. Your award, therefore, has been confirmed for the present.

Your address on our records is as shown in this letter. Should you change your address, please notify us promptly.

Your case will be followed and you will be advised when re-examination is considered necessary.

Yours very truly,

ADJUDICATION BRANCH.

  
S. Agostini,  
Claims Adjudicator.  
c.c. Cdn. Johns Manville  
DD





2 Bloor Street East  
Toronto, Ontario  
M4W 3C 3  
Telephone (416) 965- 9938

The Workmen's Compensation Board



September 15, 1976

Mr. Michael Litke  
23 Harwood Ave. N.  
R. R. #1  
Pickering, Ont.

Dear Mr. Litke:

Claim S10186035

In a telephone call to this office, Mr. Charles Neilson of your union notified us that you had been taken to hospital July 27th, 1976, with an acute chest condition. He raised the question as to whether the chest condition was the result of the condition for which you were receiving a pension in this claim.

After getting waivers from you, we have obtained the necessary medical reports, concerning your admission in July, 1976. We have consulted our medical staff regarding the interpretation of the reports, and the Claims Review Branch has studied the situation.

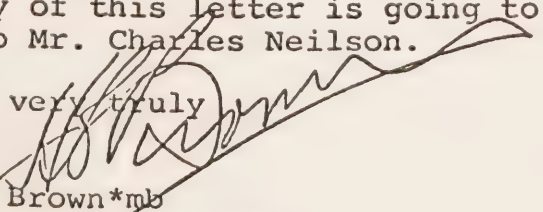
The pension you are receiving is for asbestosis, which is a fibrotic condition in the lungs resulting from exposure to asbestos over many years. The condition which took you to hospital July 27th, 1976 was diagnosed as a heart attack, of a kind which results from a degenerative condition in the blood vessels supplying the heart muscle. It would not result from your asbestosis, nor from any other disease from which you might have been suffering prior to the attack.

Entitlement for the coronary heart attack in July, 1976 and for its treatment must be denied.

The above decision is open to appeal and information on the appeals procedure may be found in the attached pamphlet.

A copy of this letter is going to Canadian Johns Manville Ltd., and to Mr. Charles Neilson.

Yours very truly

  
H. S. Brown\*mb  
Claims Review Branch  
enc. I.A.A.

When writing the Board please quote the above file number





could go on and on but the point that I am trying to bring across is the similarity to my husband's case.

Gus has severe fibrosis on both lungs with severe scarring and thickening of the pleural membranes. I was told this by the doctor in Scarborough General Hospital in March 1981. When Gus was admitted with severe breathing problems. (I am requesting these Xrays).

Each time Gus entered the hospital he was given antibiotics intravenously and put on oxygen.

Last year Gus is having severe abdominal pain. He is on very heavy medication. Because of Gus's severe breathing problems - the doctors cannot do an exploratory.

From an actual case 1980 - From the W.C.B. - Section 75

In its decision of date 1980, the Appeal Board concluded that Mrs. X is not entitled to dependency benefits because Mr. X's death did not result from his asbestosis condition.

On reconsidering the Appeal Board decision the Board concluded that the reason surgery, could not be done, was due to the presence of compensable asbestosis. The Board, therefore, finds that the ultimate cause of Mr. X's death was his compensable asbestosis condition. This again is my husband's condition.

Believe the facts speak for me.

Gus and others like him should be 100% compensated. Arthritis and other symptoms that appear could be caused by asbestos. The cases I have shown you indicate very clearly, that W.C.B. are not doing a very good job.

Needless to say, it is the asbestos victims like my husband and myself who suffer not only emotionally, but financially, I would appreciate any help that I might receive in order to win my husband's case against W.C.B.

Yours very truly,

Betty Glaser

B. Glaser





The Bendix Corporation  
Executive Offices  
Bendix Center  
Southfield, Michigan 48037

Ms. Linda Kahn  
Executive Coordinator  
Royal Commission on Matters of  
Health and Safety Arising from  
the Use of Asbestos in Ontario  
180 Dundas Street West  
22nd Floor  
Toronto, Ontario  
M5G 1Z8

June 11, 1981

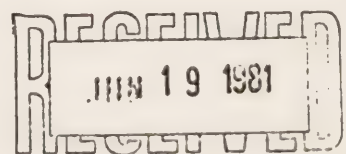
Dear Ms. Kahn:

We are in receipt of the transcripts of public hearings of the Royal Commission on Asbestos transmitted to our attention on May 28, 1981.

On March 27, 1981 we transmitted a brief "Bendix Automotive of Canada, Ltd," Presentation to the Royal Commission on Matters Arising from the Use of Asbestos in Friction Materials. We believe this document represents the factual information on our operations.

There is only one point we would like to clarify for the Commission resulting from the hearing held in Windsor on March 27, 1981. A Mr. Robert Taylor advised the Commission his doctor had advised him he had asbestos fibers in both lungs after working at Bendix Automotive of Canada, Ltd. since September 26, 1968.

Attached please find a copy of a memo from the Claims Review Branch of the Ontario Workers Compensation Board. The report indicates "In all fairness to yourself and the employer, we arranged to obtain the radiological evidence and these were reviewed by our medical advisors on chest disabilities. The X-ray was essentially a normal one with no evidence of any thickened pleura or pleural plaques. Certainly there is no evidence of any asbestos dust effects in the parenchyma of your lungs. It was the medical opinion that you do not have asbestosis".









June 11, 1981  
Page Two

In closing, we would like to thank you for transmitting the activity of the Royal Commission to our attention. If there are any specific questions you or the board may have regarding the Bendix Automotive of Canada, Ltd. operations, please advise and we will attempt to obtain the information.

Sincerely,

A handwritten signature in black ink, appearing to read "J. W. Armstrong", with a stylized flourish at the end.

J. W. Armstrong  
Corporate Director  
Safety and Protection Services

JWA/gvp  
Attach.





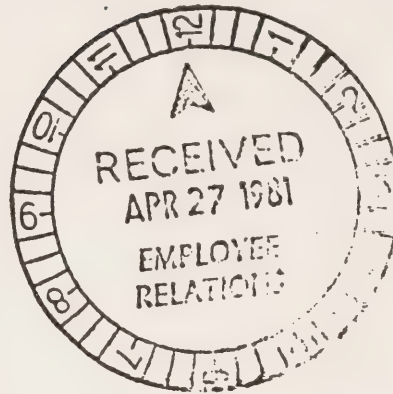
2 Bloor Street East  
Toronto, Ontario  
M4W 3C3 965-8853  
Telephone (416)

# Workmen's Compensation Board



FORM 922

Bendix Auto of Canada Limited  
945 Prince Road  
WINDSOR, Ontario  
N9C 2Z4



Claim - S13294817 - Robert Taylor.

We are enclosing for your information and records, a copy of the correspondence addressed to Mr. Robert Taylor.

*M G Falco*

M. G. Falco  
CLAIMS REVIEW BRANCH



965-8853

April 16, 1981

Mr. Robert Taylor  
1064 Brock Street  
Apt. #210  
WINDSOR, Ontario

Dear Mr. Taylor:

Claim - S13294817

On February 20, 1981, you approached our Windsor Area Office and requested a claim be established for asbestosis while working for Bendix of Canada Limited. The above claim was established and reports and information were obtained from yourself, employer, and the attending physicians involved. Your file has now been referred to the Claims Review Branch for further consideration.

Your report indicates being employed with Bendix automotive of Canada from September 26, 1968 to April 1980, and you were employed in the making of power brakes. The company report indicates that you worked in the general pool as an assembler during the period and from time to time, you worked as an inspector for short stints of 4 to 8 hours per day. Your family doctor's report suggest the disability was asbestosis of the lungs as you were exposed to asbestos inhalation while working at Bendix since September 1968, and that the x-ray reports of the chest reveal tiny spots which are most likely due to asbestos. However, the doctor's report on Occupational Chest Disease indicates that there is no acute intra-thoracic disease at present, but the presence of small, discrete, pinpoint calcifications in both lungs may be significant, when considered in the light of your history of exposure to asbestos.

In all fairness to yourself and the employer, we arranged to obtain the radiological evidence and these were reviewed by our Medical Advisors on chest disabilities. The x-ray was essentially a normal one with no evidence of any thickened pleura or pleural plaques. Certainly, there was no evidence of any asbestos dust effects in the parenchyma of your lungs. It was the medical opinion that you do not have asbestosis.

cont'd .....





cont'd ...../2

Claim - S13294817 - Robert Taylor

The Claims Review Branch has also examined all the information available. Although your family doctor has diagnosed asbestosis, his report indicates that there are no special symptoms at the present time and you are asymptomatic. The radiological evidence on which the diagnosis was based, has been reviewed by our Medical Advisor on chest diseases and no evidence of asbestosis was found. Therefore, considering the opinion of our Medical Advisor on chest diseases and the above information, the Claims Review Branch regrets that your entitlement for asbestosis has not been established.

We regret that we must deny any Workmen's Compensation benefits for lost wages or medical aid cost dealing with this disability.

The above decision is open to appeal and information on the appeals procedure may be found in the attached pamphlet.

A copy of this letter is being forwarded to your employer.

Yours very truly,

M. G. Falco  
Claims Review Branch  
Enc. I.A.A.  
/shl





we  
66

BRIEF TO THE ROYAL COMMISSION  
ON MATTERS OF HEALTH AND SAFETY  
ARISING FROM THE USE OF ASBESTOS  
IN ONTARIO

FROM:  
THE HAMILTON AREA OCCUPATIONAL  
HEALTH AND SAFETY COMMITTEE  
OF HAMILTON LABOUR COUNCIL.

See also Brief #12



In our previous brief we brought into serious question the value of yet another extensive investigation such as this Commission, at a time when action is required on the basis of the present massive accumulation of information on asbestos.

We now address the issue of compensation for asbestos related disease. The fact that we are participating in this manner in the Commission in no way reflects a lessening of our concern about its misplaced priorities. Also the fact that we will be discussing this issue within the present framework of Workers' Compensation does not imply satisfaction with the present woefully inadequate system of supporting workers at times of illness or injury.

We know personally, as well as through the experiences of others, of numerous cases of injustice at the hands of the Board suffered by workers with asbestos-related disease. We will not dwell on these, as labour has been far from silent on this issue.

In this brief, we will address the adequacy of the WCB criteria or guidelines for adjudication of claims pertaining to asbestos-related cancer. This is not intended as an exhaustive analysis of these criteria; the goal is to indicate certain of their deficiencies, and suggest corrections.





The reason that we choose this approach is that the limitations in these criteria illustrate clearly a fundamental problem with the WCB: its incapacity to apply the benefit of reasonable doubt in a fair fashion.

Before looking at these criteria more closely, we wish to make two more points. Firstly, by taking this approach we do not mean to endorse the application of such written criteria in WCB claims as a general policy. This is a large question that this brief does not deal with.

Secondly, it is widely said that actual WCB practice or policy may differ from the written guidelines presumably in the direction of compassion or leniency. If this is the case, this offers no opportunity for objective comment, and we must restrict our focus to the published criteria. In any event, once again we know of numerous cases in which workers appeared to meet the guidelines and yet were denied compensation, and we are aware of no over-generous awards.



For illustration, the guidelines for adjudication' of mesothelioma claims used by the Workman's Compensation Board are reviewed, in some detail.

Such claims are said to be favourably considered when:

1.1 There is a clear and adequate history of at least 10 years occupational exposure to asbestos.  
and

1.2 There is a minimum interval of 15 years between first exposure to asbestos and the appearance of mesothelioma.

Under other circumstances, the 'benefit of reasonable doubt' is to apply. (1)

Examination of the literature does indicate that a latent interval of less than 15 years is unusual. (2)

However, condition 1.1 is inconsistent with a well-accepted characteristic of mesothelioma: its frequent occurrence following short-term exposure. For example, McDonald and McDonald reported nine mesotheliomas in 56 deaths among 199 workers exposed to crocidolite during manufacture of gas masks, in Canada, from 1939 to 1942. (3) Jones found a high incidence of mesothelioma in women asbestos workers (5), seven of 14 mesotheliomas had less than two years of exposure. In Wagner's analysis of 33 cases (6), five had total exposure of 3 to 6 years.

In addition to its disregard for short exposure times, three other factors throw doubt on the validity of this guideline;

- i) the pervasive use of asbestos in industry
- ii) the highly specific relationship of asbestos with mesothelioma
- iii) the frequent association of mesothelioma with low intensity exposures.





The interaction of these factors is discussed in the following paragraphs.

A partial list of industrial uses of asbestos includes (7):

- heat insulation
- brake linings
- electrical wire insulation
- pipe and furnace fitting
- asbestos textiles--clothing, blankets
- vinyl asbestos tile
- air filtration
- paint filler
- shingles, asbestos boards
- pump packing
- fireproofing steel beams

Clearly, working with asbestos is not restricted to mining, milling, and manufacturing processes utilizing raw asbestos, but includes construction, insulation, repair, and demolition activities; work with the above commodities; and employment in physically adjacent circumstances (eg. carpenters, welders, ...) Thus, retrospective determination of occupational exposure, never straightforward, can be extremely difficult because of the pervasiveness of asbestos in the work environment, and the wide variety of forms, of which the worker may not be aware, that it may take. This problem complicates all epidemiologic studies in the etiology of mesothelioma.

Notwithstanding these difficulties, no other causal agent has been demonstrated. Approximately 85 % of mesotheliomas are directly attributable to asbestos. (8) Refinements in history taking and pathologic technique and expertise, as well as the advent of the electron microscope, have been in-



creasing this proportion. (9, 10, 11)

Mesothelioma is well documented to occur in association with low levels of exposure. One-third of Wagner's cases (6) had merely lived in the vicinity of asbestos mines and mills. Harries found that 53 of 55 cases in the Royal Navy shipyards occurred in those only peripherally exposed (12). Anderson (13) has identified 5 mesotheliomas among family contacts of 1,664 insulation workers.

Thus, the low levels of exposure associated with sources of asbestos which may not be obvious or understood, put many workers at risk of mesothelioma, (and other asbestos-related cancer). Conversely, any case developed will in all likelihood have been caused by an asbestos exposure.

It follows that this guideline i) is based on erroneous premises, and ii) cannot apply 'the benefit of reasonable doubt', without taking account of the above considerations. On the basis of current knowledge, in all but clearcut non-occupational cases, adjudication of mesothelioma claims should acknowledge compensable occupational asbestos exposure.





We now review the WCB criteria for lung cancer.

Unlike mesothelioma, lung cancer can be caused by many factors other than asbestos. These different relationships have made it difficult to detect epidemiologically a causal link with asbestos at relatively small levels or durations of exposure.

However there is ample evidence that exposure duration much shorter than 10 years (the WCB criteria) can cause a considerable increase in risk of lung cancer. (In fact, based on his analysis, Knox (14) has postulated that cancer risk is proportionately higher at lower dust levels or shorter exposures.)

Newhouse (15) observed, among workers "severely" exposed for less than 2 years, 20 cases of lung cancer in men when 6.6 were expected and 6 cases in women when one was expected. Similarly, Seidman (16) studied workers who were first employed in an amosite asbestos factory during the years of World War II. He found a ratio of observed to expected lung cancers, ranging from 2.24 in the category of workers exposed for less than one month, through to over 5 for those exposed between 6 and 12 months, to over 7 for those exposed for more than one year. Enterline (17), using a combined index of intensity and years of exposure, found an increased and steadily increasing number of observed as compared with



expected deaths from lung cancer, starting with the lowest exposure category.

We believe then that the literature demonstrates that short duration of exposure to asbestos certainly can lead to lung cancer.

This conclusion only reflects the general consensus in the scientific community that there is no level of exposure to a carcinogen which produces no risk.

With regard to gastrointestinal and laryngeal cancer, the required latent period, according to the WCB criteria is 20 years. For gastrointestinal cancer, the worker must have had a clear, adequate, continuous or repetitive exposure, and for laryngeal cancer the criterion states exposure duration must be 10 years.

But when we now turn to the literature to seek the basis for these criteria, we are at a loss to explain how they were arrived at. But we were also in this quandry when attempting to understand the lung cancer and mesothelioma criteria.

We must discuss briefly the nature of epidemiological research. The unique association between mesothelioma and asbestos has enabled individual cases to be followed back in time and much to be learned about their dose response relationship. Accordingly, it has emerged that minimal exposures can cause the disease.





Lung cancer is multifactorial in origin, but the fact that the relationship with asbestos is quite strong has allowed the risks of short exposures to emerge.

With gastrointestinal cancer and laryngeal cancer, either the risk although present is smaller than for the previous cancers discussed, or asbestos is not deposited in the same intensity of dose at these sites.

However we know of no sound epidemiological study which has demonstrated, with adequate sample size, that there is no increased risk of asbestos-related cancer of the gastrointestinal tract or larynx, with short exposures to asbestos, or with latent periods of less than 20 years. It may very well be that studies have been too small or improperly designed to detect such risks. We would certainly appreciate such evidence being brought to our attention if it is available.

Thus we make the following recommendation which we believe to fulfill the proper application of reasonable benefit of doubt, in accordance with foregoing:

All workers with a demonstrable history of exposure to asbestos, whether incurred in an asbestos industry



per se or in one of the many other occupational settings where one may encounter this hazard, and who have developed any of the asbestos-related cancers, should receive compensation, based on the use of the minimum known exposure periods and latent periods that have produced any of the asbestos-related cancers.

The ultimate solution to the problem of asbestos-related cancer is the abolition of asbestos from our environment.





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WC  
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A. Lever  
198 Old Colony Road  
Kanata, Ontario  
Phone: 613-836-1614  
(Home)  
Phone: 613-432-4886  
(Office--Renfrew)  
February 9, 1981

Ms. Linda Kahn  
Executive Co-ordinator  
Royal Commission on Asbestos  
180 Dundas Street West  
22nd Floor  
Toronto, Ontario  
M5G 1Z8

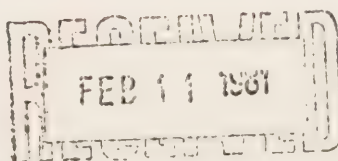
Dear Ms. Kahn

I was interested in the notice in the OTTAWA CITIZEN recently regarding the ROYAL COMMISSION ON ASBESTOS. The following is for your information, and in no way is a submission to the Commission.

I was born and raised in a textile town, Rochdale, in the industrial north of England. One of the industries in that town was a company then named TURNER BROS. I believe it subsequently became TURNER-NEWALL. While I never worked there, I know the main function was the spinning and weaving of asbestos materials. Prior to the 1939-45 war, I recall almost weekly reports of deaths from employees. I believe Coroner's Inquests were held in many cases, and the claim was that the employees died from a disease known as ASBESTOSIS. The reports were in the local newspaper named the ROCHDALE OBSERVER. I believe that paper is still published. If you were interested in approaching them for accounts of some of the things I have mentioned, I believe the address would simply be:

THE ROCHDALE OBSERVER,  
ROCHDALE,  
LANCASHIRE,  
ENGLAND.

I know the trade unions of the time were always in conflict with the employers, claiming of course, health hazards. I believe the employer those days took the stance that deaths were caused by other respiratory diseases. I left Rochdale during the war, and of course, the company may or may not





...2

exist at this time. Once again, I'm sure you will get in touch with them if you feel they can be of assistance.

This is submitted in the hope that you may be able to take advantage of research and studies already undertaken.

Sincerely

A handwritten signature in cursive script, appearing to read 'Albert Lever', with a long horizontal flourish extending to the right.

Albert Lever

mjm

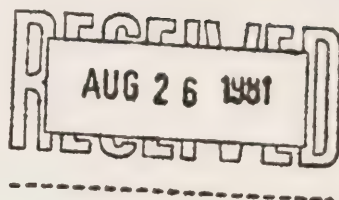




Wolfgang Pallese,  
206 Craydon Road,  
Whitby, Ontario.  
June 21, 1981.



Royal Commission  
on Matters of Health and Safety  
Arising from the Use of Asbestos in Ontario.



Sirs:

Re: Asbestos worker and  
the Workmen's Compensation Board

My experience with the Workmen's Compensation Board:

On October 26th, 1977, I was interviewed by the Workmen's Compensation Board and informed that my continuation of work at Johns-Manville was not advisable, I should "get out" and the Workmen's Compensation Board would retrain me at any job I liked, including University. Because of a divorce and incoming expenses I deferred my decision. I was advised that I could retrain at any time suitable for me.

In February 1980, Johns-Manville made me an attractive offer which would make retraining financially possible with the help from the Board. Before I accepted I telephoned Mr. W. Pearse for advice. He told me: "This is good news indeed, and by all means accept. We will have you in retraining immediately." He also wrote me a letter, dated February 27th 1980, which ended with this sentence: "I will communicate with you by the end of the current month." That never happened. I phoned him during the next two months at least forty times. Each time I was told to be home the following day, in the morning, and he would place me in retraining. My home waiting time was at least 400 hours or more!

I decided then to go to my Member of Parliament, George Ashe - but drew a blank - no response. Then, to the Leader of the Opposition, Stuart Smith, and the matter was taken up in Parliament, but again no response or positive action from the Ontario Conservative Government.



I went to the Ombudsman of Ontario and received the following answer:

"Your application for retraining has been rejected." I found that very interesting because I never filled an application, but had been told by the Compensation Board to retrain and I complied with their advice.

Because it is obvious that the Government of Ontario and their civil servants cannot be trusted in this case, I flew to Germany to get a complete medical check-up at the University Clinic in Homburg (Saarland), at the Department of Pneumology. We all know now that the reports from the Ontario Government cannot be trusted regarding the hazards of asbestosis, so here are the facts, this time not changed, rearranged or covered up, but given by unbiased doctors unrelated to this case:

	<u>Subject</u>	<u>Normal</u>
<u>1. Whole body Plethymostrophy</u>		
resistance	0.19	0.3
specific resistance	0.51	1.0
intrathorakales		
gas volume	2.69	3.8
residual volume	1.74	2.0
vital capacity	4.0	5.5
total capacity	5.74	7.5
<u>spirometry</u>		
vital capacity	4.0	5.5
one second capacity	3.0	4.0
<u>2. Blood Analysis</u>		
Oxygen partial resistance	9.74	10.5
Carbon dioxide resistance	4.77	5.2
p.h. value	7.37	7.4
actual bicarbonate	21.80	23.4

To really evaluate these tests it must be taken into consideration that I was a marathon runner with many trophies, and my original lung capacity



far exceeds any normal lungs.

If I run today, lack of oxygen occurs after two hours and my lips turn blue. Also, my heartbeat increases to an abnormal beat, but I have found that if I take pure oxygen, as in the Lifeogen emergency bottles, I can bring it to normal within a few hours.

Conclusion: I have never asked for any "hand-out", and even today I prefer to be a part of our society as a contributing member, rather than one in the welfare line-up draining Canada of its funds. But, I have been misled by the Workmen's Compensation Board and given false promises (in writing and signed), only to find myself without any security for my future or any hope for a new job. I have written many applications without results because when a company knows that I was an asbestos worker for over twenty five years they do not want to take any risks - and so I find myself unemployable. Such a situation seems to be discrimination, for which I see no escape or help.

Is it so much to ask for retraining, so that the asbestos workers can support their families, and still hold their heads up with pride, or would the Government of Ontario prefer to have us all on their welfare rolls instead of keeping us as honest tax payers? These are some questions that I would like to ask Premier Davis himself.

Sincerely,

Wolfgang Pallaske  
Wolfgang Pallaske.

WP:





2 Bloor Street East  
Toronto, Ontario  
M4W 3C3  
Telephone (416) 965-8618

The Workmen's Compensation Board



March 6, 1980

Mr. Wolfgang Palleske  
206 Craydon Road  
Whitby, Ontario  
L1N 2B6

*Copy of letter from  
compensation board.*

*I am still August 1981  
waiting*

Dear Mr. Palleske:

RE: W.C.B. CLAIM #S12397247

This will confirm our telephone conversation of February 27, 1980.

You did inform me that you now desire to participate in our Special Rehabilitation Assistance Programme. Our records show that you were interviewed in this regard on October 26, 1977 at which time you elected to defer your decision. This is of course the first time that we have heard from you from that time.

We are at the present undertaking a review of several cases with Johns-Manville and hopefully I will be in the position to communicate with you by the end of the current month.

Yours very truly,

*Wm D Pearce*

Wm. D. Pearce  
Rehabilitation Specialist  
VOCATIONAL REHABILITATION DIVISION  
WDP\*j1

cc—Mr. John Duncan, Employee Relations Manager

When writing the Board please quote the above file number



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SUBMISSION TO

THE ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY

ARISING FROM THE USE OF ASBESTOS IN ONTARIO

FROM

THE WORKMEN'S COMPENSATION BOARD, ONTARIO

JUNE 15, 1981





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## ASBESTOSIS AS AN INDUSTRIAL DISEASE

All industrial diseases giving rise to disability are covered either under:

- Section 1.-(1)(a)(iii) - "disablement arising out of and in the course of employment,"
- Section 1.-(1)(L) - " 'industrial disease' means any of the diseases mentioned in Schedule 3 and any other disease peculiar to or characteristic of a particular industrial process, trade or occupation,"
- Section 118,

or

- by Schedule 3 of the Act which is a list of industrial diseases closely allied to specific industrial processes and in which the relationship between the disease and a specific employment is clearly established and can be presumed almost automatically.

The original Workmen's Compensation Act in 1915 provided coverage under Schedule 3 for only six industrial diseases. Coverage for asbestosis became possible in 1926 when "pneumoconiosis" was added to Schedule 3 by amendment to Regulation 94.

The first claim for asbestosis was allowed in 1942 and a total of 119 claims for asbestosis were allowed up to 1975, including some where mesothelioma was also present.

### MEDICAL GUIDELINES FOR DIAGNOSIS OF ASBESTOS FIBRE DUST EFFECTS (AFDE) (approved by the Board on May 11, 1976)

#### 1.0 Background

1.1 Historical: The concept and the practical application of the Mineral Dust Effect (MDE) have no precedents established in any known compensation jurisdiction and the terms "pre-silicosis" or "pre-asbestosis" are not generally found in the medical lexicon or used with any consistency by investigators or expert panels or discussants at seminars.

In the voluminous publication of the papers presented at the International Conference on Pneumoconiosis at Johannesburg in 1969, the term "pre-asbestosis" is mentioned only once and without comment. However, it is clear that throughout the conference a concern was in evidence over the continuing inability to monitor and prevent severe disease in dust occupations. There was general implied agreement on the need to deal with dust disease at an earlier stage than was generally in evidence.



Dr. W.J. Smither - (Reference 4A) stated that - "a working party is now being set up to reconsider the diagnosis of pneumoconiosis. We hope to influence the legal powers in such a way as to give great statutory strength to our attempts to certify and protect from further exposure the early case of asbestosis. The period between the first x-ray change and certification is far too long."

1.2 Clinical: The concept of the MDE may be applied to both particulate and fibrous minerals, although in the definition and evaluation of each the criteria are dissimilar and the presumptions used in one cannot be used in the other.

For example, to establish the Silica Particle Dust Effect (SPDE), one needs only to identify the following.

1.2.1. - a characteristic x-ray.

1.2.2. - adequate history of exposure.

In contrast, no characteristic x-ray exists in the AFDE and an adequate history of exposure must be supplemented by the known absence of significant functional changes as reflected in the Pulmonary Function Studies (PFS) and clinical examination.

Again, in SPDE (code 4) x-ray and observer consistency are easily achieved - not so with AFDE. In AFDE, early x-ray signs are often undefinable and quite variable. There may be marked differences in the prevalence of linear changes, pleural thickening, or calcification in persons at risk in milling, mining or fabrication of asbestos operations. Pleural changes for example are often the predominant early manifestation of AFDE in insulators, while in fabrication operations the early changes are parenchymal in the form of coarse linearity at the bases adjacent to the diaphragm. Such early changes as these are usually present without abnormal PFS.

The pleural reaction is probably the earliest x-ray sign of AFDE, either in the form of variable costophrenic sulcus obliteration or discrete (focal) noncalcific pleural thickening.

### 1.3 Radiological and Physiological:

1.3.1. - SPDE - the fibrotic or reticular changes in SPDE:

1.3.1.1. - are punctate or nodular.

1.3.1.2. - are not interstitial.

1.3.1.3. - are discrete and reflect x-rays in an identifiable pattern.

1.3.1.4. - are not accompanied by pulmonary function impairment.



1.3.1.5. - do not obliterate the vascular pattern.

1.3.1.6. - do not produce pleural thickening,

#### In Contrast

1.3.2. - AFDE - with respect to the fibrotic or reticular changes:

1.3.2.1. - are largely interstitial rather than nodular,

1.3.2.2. - the pulmonary vascular pattern may be distorted,

1.3.2.3. - physiological effects may be present, although of a low order.

1.4 Histological: The earliest asbestos fibre effect involves the the respiratory bronchioles and the associated alveoli by way of collagen-poor fibrosis or merely reticulin proliferation. Such changes may produce some reduction in lung compliance and in the number of functioning alveoli, although generally such changes do not affect the vital capacity or diffusion rate.

By the time radiological manifestations are visible such tissue changes may have progressed to involve the most distal alveoli in the lower lung fields.

Therefore, it must be assumed that in AFDE a wide spectrum of structural change will be present, e.g. from simple reticulin proliferation to frank collagen-rich fibrosis. Such change while not extensive, will likely be demonstrated on x-ray examination and be confined to the basal lung tissues.

## 2.0 Identification of The AFDE Requirements

Identification presupposes that structural alterations have not quite advanced to a level which produce significant physiological impairment.

Therefore, we need to identify those x-ray signs and/or functional changes known to exist in asbestos workers which apparently cause no significant work impairment but which in the past are known to have preceded progression and development of subsequent impairment. There is no certainty that removal from exposure will prevent progression of changes and there are no means of identifying those persons at risk who will progress, but it seems reasonable to assume that the earlier the removal the less will be the chance of progression.

## 3.0 Removal From Risk is Not Indicated Under Any of The Following Circumstances:

3.1. - pleural reaction is minimal and exists alone.

3.2. - basilar parenchymal, changes exist alone.





- 3.3. - serial x-rays have not changed for 5 to 10 years.
- 3.4. - serial studies of vital capacity show no change.
- 3.5. - chest x-ray changes do not seem compatible with the exposure history.
- 3.6. - exposure is less than 10 years.
- 3.7. - exposure is in excess of 30 years showing minimal or borderline changes as above.

#### 4.0 Acceptance Criteria - AFDE

It is recommended that a case be accepted as showing AFDE when the following circumstances apply.

- 4.1. - an adequate history of exposure is documented - a minimum of 10 years unless an unusual intensity exposure is established.
- 4.2. - when at least two of the following radiological signs are present.
  - 4.2.1. - intralobar pleural thickening (major or minor fissures).
  - 4.2.2. - variable obliteration of the costophrenic angles.
  - 4.2.3. - variable pleural thickening of a focal or plaque-like nature along the lateral chest wall or diaphragm is noted.
- 4.3. - And when in addition at least two of the further following radiological signs are present.
  - 4.3.1. - horizontal linear markings 1.3 mm. thickness in the lower zones usually bilateral.
  - 4.3.2. - a general coarsening of the lower zone linear pattern with partial replacement by a reticular or net-like pattern.
  - 4.3.3. - superimposed of minute bead-like opacities. 1-2 mm. in diameter over or adjacent to the lower zone pulmonary arterial tree.
- 4.4. - these changes described above must have occurred during the preceding 5 to 10 year period.
- 4.5. - the vital capacity studies may have shown changes over the past 5 years although the last result is still within normal limits.



REFERENCES

(1) Occupational Lung Disorders

W. Raymond Parkes, 1974

(2) Occupational Lung Diseases

Morgan and Seaton, 1975

(3) Biological Effects of Asbestos

Annals of the New York Academy of Sciences  
Volume 132, P. 1-776, 1965

a) Asbestos in Great Britain  
(J.C. McVittie)

b) Routine Pulmonary Studies on 380 Employees of an  
Asbestos Process  
(R. Hunt)

(4) Pneumoconiosis

Proceedings of the International Conference -  
Johannesburg, 1969

a) Symposium on the Radiology of Asbestos.



## PROCEDURAL GUIDELINES FOR CLAIMS ADJUDICATORS

### - ASBESTOSIS -

Asbestosis was included in Schedule III on April 8, 1926 and is accepted under Sections 1-(1) (L) and 118 of the Ontario Workmen's Compensation Act as peculiar to and characteristic of a process, trade or occupation involving exposure to asbestos.

Reference: - Sections 1-(1) (L) and 118 of the Act;  
- Board Orders and Administrative Directives,  
pages 192 to 194.

#### Requirements for Consideration of Allowance

It must be established that:

1. There is a clear and adequate history of occupational exposure to asbestos;
2. There is a diagnosis of frank asbestosis;
3. Advisory Committee (A/C) of the Ministry of Labour has examined referred cases of occupational chest disabilities and submitted a detailed report of its findings with recommendation on the degree of functional impairment.

#### Procedural Guidelines

1. Send Employee's Report of Occupational Disease, Form 6S, to worker; Employer's Report of Occupational Disease, Form 7S, to employer; Doctor's Report of Occupational Disease, Form 8S, to attending doctor. Send "Mining History Memo", where applicable, to Statistical Branch.
2. Approved follow-up and jacket mark-up procedures apply.
3. In cases where normal enquiries do not provide sufficient information, arrange for an investigation by directing a memo through the Team Co-ordinator.





4. Refer claim with a memo to Consultant, Chest Disease, through the Team Co-ordinator, via the Industrial Disease Specialist. Include the following:

- Mark-up "A/C Memo" inside of jacket;
- Use A/C stamp to identify copies of reports for Advisory Committee;
- Industrial Disease Specialist completes Form 374 and directs a memo re appointment to Consultant, Chest Disease.

5. No entitlement

Check whether the Advisory Committee is arranging follow-up examinations. If so, the Adjudicator sends Form 1513 (SC/3) to the worker, copy to the employer, and to other representatives, where applicable.

If no follow-up indicated, direct Form 899 to the Claims Review Branch, through the Team Co-ordinator, with recommendation for denial. (Usually G2(4), G2(5)).

6. Entitlement

- 6.1 Notification re Allowance for Medical Aid

If allowance for "medical aid only", a detailed letter is sent to the worker, copies for the employer and other representatives (if applicable) outlining the allowance.

- 6.2 Notification of Pension

Send SC7 to worker, copy for the employer and other representatives (if applicable).

7. Subsequent Advisory Committee Reviews

Once a subsequent Report is received from the Advisory Committee, determine if the condition has changed. If so, send Form SC7 advising employee claim allowed for pension. In cases where pension has increased, send Form SC9, if increase was to 100%, also send Form 0904.

If A/C still indicates no chest condition, send a subsequent SC3. If A/C indicates no change in award, send SC6 to worker.

8. Charging of Costs

S.I.E.F. relief does not apply to cancer and chest disabilities.

If there is more than one exposure employer, the costs of the claim are charged to the last exposure employer. For example, if the worker had been employed:

- 5 years with employer "A" (1945 - 1950)
- 10 years with employer "B" (1950 - 1960)
- 10 years with employer "C" (1960 - 1970)
- 9 years with employer "D" (1970 - 1979)



the costs of the claim would be charged to employer "D" - the last exposure employer.

## 9. Benefits

Claims submitted for cancers and chest disabilities usually result in permanent disability award, but on occasion temporary total and temporary partial difference payments will apply.

### 9.1 Permanent Disability Wage Basis

- If the worker was in exposure employment at the time the diagnosis was made, (or an earlier date, if approved by the Board's Consultant, Chest Disease) the earnings obtained are for the year immediately prior to the date of diagnosis.
- If the worker is no longer employed in exposure, then comparable earnings of those of a worker working for the same employer, doing the same job, are obtained for the year immediately prior to the date of diagnosis.

### 9.2 Temporary Total Benefit

- If active treatment is taking place
- Same basis is used as for permanent disability, (see 9.1)

### 9.3 Temporary Partial Difference Benefits

- If the worker is requested to change employment (this must be approved by the Board's Consultant, Chest Disease), entitlement exists for payment if there is a wage loss.
- Consideration should be given to entitlement under the Board's Special Rehabilitation Programme To Remove Employees From The Hazards of Industrially Generated Diseases.
- The permanent disability wage basis is compared with the worker's present wage basis.
- Payment is granted until no difference exists or a pension is awarded.

### 9.4 Dependency Benefits

- In case of death, consider whether the cause was due to asbestosis.
- Send a memo with a recommendation to Consultant, Chest Disease.
- See "Fatal Claims" for awards payable.

NOTE: For copies of the report forms, etc., mentioned in these guidelines, please refer to Appendix 7.



CLAIMS ALLOWED FOR ASBESTOSIS, SILICO-ASBESTOSIS OR MESOTHELIOMA - 1942 - 1980  
(excluding allowed Claims for AFDE)

MESOTHELIOMA

Year Claim First Allowed	No. of Claims	Name of Last Exposure Employer	MESOTHELIOMA		Number of Deaths	DEATH BENEFITS NOT ALLOWED	
			With Asbestosis	Without Asbestosis		Rejected No. of Claims	O/S* No. of Claims
1942	1	Cdn. Johns Manville Co. Ltd.			1		
1947	1	Raybestos-Manhattan (Can.) Ltd.			1		
1948	1	Raybestos-Manhattan (Can.) Ltd.			1		
1952	1	Vincent Cutler Insulation Ltd.			1		
1956	1	Raybestos-Manhattan (Can.) Ltd.			1		
1958	1	Cdn. Johns Manville Co. Ltd.			1		
1961	1	Cdn. Johns Manville Co. Ltd.			1		
1962	1	Raybestos-Manhattan (Can.) Ltd.			1		
1965	1	Holmes Insulation Ltd.			1		
1967	6	Cdn. Johns Manville (2)			1	1	Peritonitis due to Cirrhosis of Liver, Cardiac Pulmonary failure
		Holmes Insulations Ltd. (1)			1		
		Canadian Asbestos Company (1)			1		
		Dept. of Public Works (1)			1		
		Raybestos-Manhattan (Can.) Ltd. (1)			1		
1968	3	Foster Wheeler Co. Ltd. (1)			1		
		Hiram Walker & Sons (1)			1		
		Cdn. Johns Manville (1)					
1969	6	White and Greer Co. Ltd. (1)					
		Bremco Mechanical Insulation (1)					
		Applied Insulation Co. Ltd. (1)					
		University of Toronto (1)	1		1		
		Raybestos-Manhattan Can. Ltd. (1)			1		
		Consolidated Plant Maintenance Co. (1)			1		
1970	12	Cdn. Johns Manville Co. Ltd. (8)	1	1	5	1	Myocardiac infarction. Silicosis.
		Schreiber Brothers Ltd. (1)					
		Armstrong Contracting Co. (1)		1			
		Toronto, Hamilton & Buffalo Rlwy. Co. (1)			1		
		Raybestos Manhattan (Can.) Ltd. (1)			1		





MESOTHELIOMA

DEATH BENEFITS NOT ALLOWED

Year Claim First Allowed	No. of Claims	Name of Last Exposure Employer	Without Asbestosis		Number of Deaths	Rejected		O/S*
			With Asbestosis	Asbestosis		No. of Claims	Cause of Death	
1971	9	Black & McDonald (1) Federal Insulation Ltd. (1) Holmes Insulation Ltd. (1) Bryant Insulation (1) Schaefer Townsend Ltd. (1) Cdn. Johns Manville Co. Ltd. (4)	1	1	1 1 1 1 1 2	1	Broncho- Pneumonia	
1972	12	Cdn. Johns Manville Co. Ltd. (5) Holmes Insulation Ltd. (2) Raybestos Manhattan Can. Ltd. (1) Asbestos Covering Co. Ltd. (1) Terminal Insulation Ltd. (1) Applied Insulation Co. Ltd. (1) Master Insulation Co. Ltd. (1)			3 1 1			
1973	24	Cdn. Johns Manville Co. Ltd. (12) Holmes Insulation Ltd. (2) Preston Mines Ltd. (1) Ontario Insulation (Oshawa) Ltd. (1) Ontario Hydro (1) Nor. Ont. of Canada Ltd. (1) Dept. of Public Works-Provincial Gov't. (1) Dewar Insulations Inc. (1) Kimberly-Clark of Canada Ltd. (1) Commerical Industrial Insulations Ltd. (1) Raybestos-Manhattan (Can.) Ltd. (1) Cem-Al Spray Ltd. (1)	3 1	1	8 1 1 1 1 1 1 1 1 1	1	Carcinoma of Prostate Broncho Pneumonia	1 10 1
1974	16	Cdn. Johns Manville Co. Ltd. (7) Lakehead Insulation Co. (1) Costs Charged to Firm #099999 (1) Dominion Foundries & Steel Ltd. (1) White & Greer Co. (2) A.C.& S. Contracting (1)	2	1	5 1 1 1 1 1	1	Anemia Carcinoma of the Prostate	1



# MESOTHELIOMA

## DEATH BENEFITS NOT ALLOWED

Year Claim First Allowed	No. of Claims	Name of Last Exposure Employer	With Asbestosis	Without Asbestosis	Number of Deaths	Rejected		O/S <sup>*</sup> No. of Claims
						No. of Claims	Cause of Death	
1974 (Cont'd)					1			
		Applied Insulation (1) Superior Insulation (1) Atlas Asbestos Co. Ltd. (1)				1	Broncho Pneumonia	
1975	43	Cdn. Johns Manville Co. Ltd. (34)	1	1	9	2	1. Gangerene of large bowel 2. Coronary occlusion due to arterioscler- osis	
		Applied Insulations (1) Port Arthur Shipbuilding Co. (1) A.C. & S. Contracting Ltd. (1) Lakehead Insulation & Plastic Ltd. (1) Abex Ltd. (1) Avro Aircraft (1) Cassid Catering Co. (1) Dewar Insulations (1) Holmes Insulations Ltd. (1)			1 1 1 1			
1976	26	Cdn. Johns Manville Co. Ltd. (14)	1	1	3	2	1. Intestinal obstruction due to thrombosis 2. Intestinal obstruction	
		E.B. Eddy Forest Products Ltd. (1) Cyanamid of Canada (1) Raybestos-Manhattan (1) Hamilton Porcelains Ltd. (1) Collingwood Shipyards (1) Lewis Insulations Ltd. (1) White & Greer Co. Ltd. (1) N.A. Smith & Co. Ltd. (Nasco Div.) (1) C.H. Hildebrandt Insulation Ltd. (1) Hamilton Match Plate (1) Atlas Asbestos (1) Babcock & Wilcox Can. Ltd. (1)		1 1 1 1 1 1 1 1 1	1 1 1 1 1 1 1 1 1			

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Year Claim First Allowed	No. of Claims	Name of Last Exposure Employer	MESOTHELIOMA				DEATH BENEFITS NOT ALLOWED	
			With Asbestosis	Without Asbestosis	Number of Deaths	Rejected		
						No. of Claims	Cause of Death	
1980	28	Cdn. Johns Manville (5) Applied Insulation Co. Ltd. (1) Sunnybrook Hospital (1) Holmes Foundry Ltd. (1) Canada Steamship Lines (1) Gov't. of Canada (Labour Canada Div.) (1) N.M. Peterson & Sons Ltd. (Steamship Div.) (1) McBrien Insulation Co. (1) Texaco Canada Inc. (1) Ontario Hydro (2) Tweed Steel Wks. (1) Standard Prods. Cda. Ltd. (1) Dewar Insulation (1) Ford Motor Co. (1) The Algoma Steel Corp. Ltd. (1) Kustoms Insulation Co. (1) Sunbeam Appliance Co. (Toronto) (1) Tri-cept Insulation (1) Kirk-Hurst Industries Ltd. (1) Atlas Asbestos (1) C.I.L. Inc. (1) Industrial Insulation (1) R.G. Waters Co. (1)		2 1 1 1 1 1 1 1  1  1  1  1  1 1 1	1 1  1 1 1  1  1  1			
TOTAL	258		10	36	108	12	3*	

\* 3 claims counted as allowed PD previously are now fatal o/s.



258 ALLOWED ASBESTOSIS CLAIMS AS AT DECEMBER 31, 1980

Year		Broad Category of Exposure					Total
Set Up	Allowed	Manuf.	Constr.	W'housing	Mntnce.	Mining	
1942	1942	1					1
1947	1947	1					1
1948	1948	1					1
1952	1952		1				1
1955	1956	1					1
1958	1958	1					1
1961	1961	1					
1961	1962	1					
1961	1974	1					
1961	1973					1	4
1964	1965	1					1
1965	1967	1					1
1966	1967	1					
1966	1973	1					2
1967	1967		1				
1967	1968	1					
1967	1967		1				
1967	1967	1					
1967	1967	1					
1967	1970		1				6
1968	1968				1		
1968	1969		1				
1968	1968	1					
1968	1969		1				4
1969	1969		1				
1969	1969				1		
1969	1969	1					
1969	1969		1				
1969	1970		1				5
1970	1970	1					
1970	1970				1		
1970	1970		1				
1970	1971		1				
1970	1970	1					
1970	1970	1					
1970	1970	1					
1970	1970	1					8

Total carried forward....37



Year		Broad Category of Exposure					Total
Set Up	Allowed	Manuf.	Constr.	W'housing	Mntnce.	Mining	
							Total brought forward..... 37
1970	1974		1				
1970	1970	1					
1970	1970	1					
1970	1970	1					
1970	1971	1					
1970	1971	1					
1970	1971		1				7
1971	1973	1					
1971	1971	1					
1971	1971	1					
1971	1971	1					
1971	1971		1				
1971	1971		1				
1971	1973		1				
1971	1972	1					
1971	1975		1				
1971	1973				1		
1971	1972	1					
1971	1972	1					12
1972	1972	1					
1972	1972	1					
1972	1973			1			
1972	1972		1				
1972	1972	1					
1972	1972	1					
1972	1972		1				
1972	1973		1				
1972	1972		1				
1972	1973	1					
1972	1973		1				
1972	1973				1		14
1973	1973	1					
1973	1973	1					
1973	1973	1					
1973	1973	1					
1973	1973	1					
1973	1973		1				
1973	1973	1					
1973	1973		1				
1973	1974				1		
1973	1976	1					
1973	1973	1					
1973	1973	1					
1973	1973		1				13

Total carried forward... 83





Year		Broad Category of Exposure					
Set Up	Allowed	Manuf.	Constr.	W'housing	Mntnce.	Mining	Total
		Total brought forward..... 83					
1973	1974	1					
1973	1973	1					
1973	1973	1					
1973	1974	1					
1973	1974	1					
1973	1973	1					
1973	1974	1					
1973	1975	1					
1973	1974				1		
1973	1974		1				
1973	1974		1				
1973	1974		1				12
1974	1974	1					
1974	1974		1				
1974	1975		1				
1974	1974		1				
1974	1974	1					
1974	1974	1					
1974	1975	1					
1974	1976	1					
1974	1975	1					
1974	1975	1					
1974	1975	1					
1974	1976	1					
1974	1975	1					
1974	1975	1					
1974	1975	1					
1974	1975	1					
1974	1975	1					
1974	1977	1					
1974	1975	1					
1974	1975	1					
1974	1975	1					
1974	1975		1				
1974	1975	1					23
1975	1975	1					
1975	1975	1					
1975	1975	1					
1975	1975	1					
1975	1975	1					
1975	1975	1					
1975	1975	1					
1975	1975	1					
1975	1975	1					
1975	1975	1					
1975	1975	1					11
1975	1975	1					
Total carried forward... 129							



Year		Broad Category of Exposure					Total
Set Up	Allowed	Manuf.	Constr.	W'housing	Mntnce,	Mining	
Total brought forward,.....							129
1975	1980	1					
1975	1976	1					
1975	1975	1					
1975	1979	1					
1975	1976	1					
1975	1978	1					
1975	1975	1					
1975	1975	1					
1975	1976					1	
1975	1975	1					
1975	1977	1					
1975	1979	1					
1975	1975					1	
1975	1975	1					
1975	1975	1					
1975	1975	1					
1975	1978	1					
1975	1977	1					
1975	1976	1					
1975	1975	1					
1975	1976	1					
1975	1975	1					
1975	1976	1					
1975	1976	1					
1975	1975		1				
1975	1975	1					
1975	1976	1					
1975	1980	1					
1975	1979	1					
1975	1976	1					
1975	1976	1					
1975	1976	1					
1975	1975	1					
1975	1978	1					
1975	1975	1					
1975	1979	1					
1975	1975	1					
1975	1979	1					
1975	1976	1					
1975	1975	1					
1975	1976		1				
1975	1979	1					
1975	1976		1				
1975	1976	1					
1975	1976		1				
1975	1979		1				
1975	1976	1					
1975	1976	1					



Year		Broad Category of Exposure					Total
Set Up	Allowed	Manuf.	Constr.	W'housing	Mntnce.	Mining	
Total brought forward.....							177
1975	1976			1			
1975	1976	1					
1975	1976	1					3
1976	1976	1					
1976	1978	1					
1976	1979	1					
1976	1976	1					
1976	1977	1					
1976	1977				1		
1976	1977	1					
1976	1977	1					
1976	1978	1					
1976	1977	1					
1976	1977		1				
1976	1977	1					12
1977	1977	1					
1977	1977				1		
1977	1977	1					
1977	1977				1		
1977	1978		1				
1977	1977	1					
1977	1977		1				
1977	1978		1				
1977	1977				1		
1977	1977		1				
1977	1977				1		
1977	1979	1					
1977	1978		1				
1977	1978	1					
1977	1978	1					
1977	1978	1					
1977	1978	1					
1977	1978	1					
1977	1978	1					
1977	1978		1				20
1978	1978	1					
1978	1978	1					
1978	1978	1					
1978	1979	1					
1978	1979	1					
1978	1979	1					
1978	1979				1		
1978	1979	1					
1978	1980		1				
1978	1979	1					10
Total carried forward							222





Year		Broad Category of Exposure					Total
Set Up	Allowed	Manuf.	Constr.	W'housing	Mntnce.	Mining	
							Total brought forward..... 222
1979	1980				1		
1979	1979				1		
1979	1979	1					
1979	1979	1					
1979	1979	1					
1979	1979		1				
1979	1979				1		
1979	1979	1					
1979	1980	1					
1979	1979	1					
1979	1979				1		
1979	1980				1		
1979	1980	1					
1979	1979				1		
1979	1979	1					
1979	1980				1		
1979	1980		1				
1979	1980	1					
1979	1980	1					
1979	1980	1					
1979	1980				1		
1979	1980				1		
1979	1980	1					
1979	1980		1				24
1980	1980	1					
1980	1980	1					
1980	1980		1				
1980	1980	1					
1980	1980		1				
1980	1980	1			1		
1980	1980	1					
1980	1980		1				
1980	1980		1				
1980	1980	1					12
GRAND TOTAL OF ALLOWED ASBESTOSIS CLAIMS BY CATEGORY OF EXPOSURE							
		<u>Manuf.</u>	<u>Constr.</u>	<u>W'housing</u>	<u>Mntnce.</u>	<u>Mining</u>	<u>Total</u>
		177	52	3	23	3	258

Source: W.C.B., P.&S.S.  
February 17, 1981.



CLAIMS ALLOWED FOR ASBESTOSIS OR MESOTHELIOMA

Report 52-1

CANADIAN JOHNS MANVILLE

1942 - 1980

Year Claim 1st Allowed	Number of Claims	Asbestosis Only	Mesothelioma Only	1942 - 1980		Current % of P.D.
				Both	Current Status Alive Dead	
1942	1	1			1	
1958	1	1			1	
1961	1	1			1	
1967	2	2			1	1 X 100%
1968	1	1			1	
1970	8	6	1	1	2 6	1 X 100%
1971	4	4				1 X 75%
1972	5	5			1 3	1 X 100%
					2 3	1 X 75%
1973	12	8	1	3	3	1 X 30%
					9	1 X 30%
						1 X 40%
						1 X 100%
1974	7	5		2	2	1 X 25%
					5	1 X 40%
1975	34	32	1	1	23 11	1 - MA
						2 X 10%
						3 X 15%
						8 X 20%
						1 X 25%
						4 X 30%
						2 X 40%
						2 X 50%



## CANADIAN JOHNS MANVILLE

1942 - 1980

Year Claims 1st Allowed	Number of Claims	Asbestosis Only	Mesothelioma Only	Both	Alive	Dead	Current % of P.D.
1976	14	13	1		9	5	3 X 10% 3 X 20% 2 X 40% 1 X 30%
1977	9	9			8	1	5 X 10% 3 X 20%
1978	11	10	1		7	4	2 - M.A., 4 X 10% 1 X 20%
1979	9	9			9		1 - M.A., 7 X 10% 1 X 20%
1980	5	3	2		4	1	1 - M.A., 1 X 10% 1 X 20% 1 - COMP.
TOTAL	124	110	7	7	71	53	

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Program Planning & Statistical Services  
February 11, 1981.





## LUNG CANCER IN ASBESTOS WORKERS

### INTRODUCTION

In 1976 the Board established a subcommittee to prepare guidelines for the adjudication of claims for various types of cancer.

In 1975 Dr. A. C. Ritchie, Professor of Pathology, University of Toronto, performed a study (Interim Report - Asbestos Disease, 1975; Appendix 2) the findings of which as well as the opinion of Dr. M. R. Becklake of McGill University (Appendix 1) and other findings were reviewed by the staff of the Board's Medical Services Division and subsequently referred to the subcommittee for the formulation of acceptance criteria for various types of cancer including cancer associated with exposure to asbestos.

The proposed guidelines for lung cancer in asbestos workers were approved by the Board on April 13, 1976.

### DISCUSSION

#### 1.0 Background

The use of asbestos increased tremendously after World War II, resulting in more claims for asbestosis in asbestos workers being reported to the Board. Claims for asbestosis were accepted where prolonged exposure to asbestos was established as were cases of lung cancer in workers suffering also from asbestosis. However, it was not considered that lung cancer in an asbestos worker could be accepted without the presence of asbestosis.

#### 2.0 Findings, up to April 1976

A definite study was performed by Dr. A. C. Ritchie in 1975 which indicated that he believed that there was no formula developed which would make duration and fibre count meaningful. In this the Board's Consultant of Chest Diseases agreed with Dr. Ritchie because the literature contained no studies comparing various suggested fibre count and duration exposure formulae against incidences of asbestosis in lung cancer.

Dr. Becklake in her letter of December 19, 1974 to Dr. C. C. Gray suggested such a formula, but it could not be considered because no fibre count was available in asbestos workers in Ontario. A formula of this nature could, maybe, have some application in the Quebec Asbestos Mines set-up, but since industrial exposure to asbestos is more hazardous than mining exposure, such a figure as Dr. Becklake presented was considered to be even more unsuitable to the Ontario situation. Despite this, Dr. Becklake had obviously taken a strong position, with respect to the benefit of the doubt, in this area.



Dr. Ritchie's studies indicated that there is an increase in the incidence of bronchogenic carcinoma in workers exposed to asbestos even though there may not be any evidence of asbestosis. This cancer usually develops in asbestos workers with over 15 years of exposure and Professor Ritchie's study left no doubt that there is an increase of lung cancer in asbestos workers.

Dr. Ritchie suggested that the acceptance criteria should be based on two presumptions.

- a) a clear history of occupational exposure.
- b) an arbitrary figure of 15 years as a reasonable minimum interval between 1st exposure to asbestos and appearance of the lung cancer.

Obviously, he felt that this 15 year interval is not firm and that some latitude should be given depending on the intensity of the exposure and where it had been very high, a shorter latent period should be considered.

#### LUNG CANCER IN ASBESTOS WORKERS GUIDELINES FOR ADJUDICATION

1. That lung cancer in asbestos workers be accepted as an industrial disease under Section 118 and Section 1(1)(L) of the Act as peculiar to and characteristic of a process, trade or occupation involving exposure to asbestos.
2. That based on medical studies, lung cancer claims be favourably considered when the following circumstances apply.
  - 2.1 There is a clear and adequate history of at least ten years occupational exposure to asbestos.
  - 2.2 There is a minimum interval of ten years between first exposure to asbestos and the appearance of lung cancer.
  - 2.3 Claims which do not meet the guidelines in 2.1 and 2.2 should be individually judged on their own merit having regard to the intensity of exposure and other factors peculiar to the individual case. The benefit of reasonable doubt applies.

Note: These guidelines do not apply to mesothelioma claims.

APPROVED BY THE BOARD

April 13, 1976



MESOTHELIOMA  
IN ASBESTOS WORKERS

1.0 Background Data up to April 1976

In some of the United Kingdom studies 85 to 90% of cases are said to be related to asbestos exposure. The latent periods seem to run between 20 and 40 years with most cases close to the higher figure. Other investigators have reported about 50% of those with mesotheliomata have had asbestos exposure. The differences are plainly of geographic and industrial origin, and by far the highest incidences of mesothelioma are seen in the manufacturing and the use of asbestos fiber rather than in mining and milling operations.

In the Ontario experience the range of exposures ran from less than 10 years to 43 years and in some of the lighter exposures, it was difficult to be accurate as to the total duration in years. The latency periods ran from 17 to 44 years.

- Average exposure - approximately 24 years

- Average latent period - 27 years

Fifty-eight percent of the cases arose out of the insulation industries while forty-two percent arose out of exposure to asbestos and silica as seen in the Canadian Johns-Manville plant in Scarborough. At least fifty percent of the cases had asbestosis in addition to mesothelioma. The average age of death was 52 years.

Risks increase as the particulate concentration and duration of exposure to asbestos increase. Smoking does not seem to increase the danger, presumably, due to the fact that this tumor does not involve lung substance.

So far, there is no evidence that mesothelioma is increased in persons not exposed occupationally. Members of the employee's family may be at slight risk.

2.0 Characteristics

Characteristically, exposure to asbestos has often been described as intermittent rather than continuous. It is accepted that heavy exposure of a matter of months may in some instances give rise to mesothelioma 20 to 40 years later. With the limited number of cases in Ontario, the average latent period is 27 years - the same as the average latent period for lung cancer, although the average age at death of the workers with mesothelioma is 10 years less than the case with lung cancer.





### 3.0 Studies Considered in the Development of the Guidelines

The following references were considered:

- 1) Dr. A. C. Ritchie's report of March 24, 1975 dealing extensively with the risks of asbestos inhalation. (Appendix 2)
- 2) Occupational Lung Disorders - by W. Raymond Parks, (1974)
- 3) Occupational Lung Diseases - by Morgan and Seaton, (1975)

Mesothelioma of the pleura or peritoneum was once considered rare, but is now recognized as a well defined neoplasm. A high proportion of such mesotheliomata are associated with a history or other evidence of exposure to asbestos.

Some investigators have reported that asbestosis is always evident in the lung of the patients with mesotheliomata. This is not the experience of all clinicians and is not the Board's experience in Ontario.

It is generally agreed that mesothelioma rarely develops until 15 to 20 years, or more, after exposure to asbestos, although cases occurring earlier have been reported. It is believed that Dr. Selikoff has reported one case in which the latency period was 13 years or so.

#### MESOTHELIOMA IN ASBESTOS WORKERS GUIDELINES FOR ADJUDICATION

1. That mesothelioma in asbestos workers be accepted under Section 118 and 1 (1) (L) of the Act as peculiar to and characteristic of a process, trade or occupation involving exposure to asbestos.

That based on medical studies, mesothelioma claims be favourably considered when the following circumstances apply:

- 1.1 There is a clear and adequate history of at least 10 years occupational exposure to asbestos.

and -

- 1.2 There is a minimum interval of 15 years between first exposure to asbestos and the appearance of mesothelioma.

- 1.3 Claims which do not meet the guidelines in 1.1 and 1.2 should be individually judged on their own merit having regard to the intensity of exposure and other factors peculiar to the individual case. Consideration will be given where it seems evident that the mesothelioma cancer resulted from occupational exposure to asbestos. The benefit of reasonable doubt applies.

APPROVED BY THE BOARD

April 13, 1976



## CANCER OF THE GASTROINTESTINAL TRACT IN ASBESTOS WORKERS

### INTRODUCTION

In 1976, the Board established a subcommittee to prepare guidelines for the adjudication of claims for various types of cancers, including gastrointestinal cancer (esophagus, stomach, small bowel, colon and rectum) in asbestos workers.

Dr. A.C. Ritchie, Professor of Pathology, University of Toronto, prepared a report, and in view of the importance of any policy to be formulated on the subject, Dr. A.B. Miller, an eminent epidemiologist, was commissioned to perform a further study.

The staff of the Board's Medical Services Division assembled and reviewed literature and data available on asbestos induced diseases, and referred all this material as well as the two independent reports to the subcommittee for the development of the adjudication guidelines.

The proposed guidelines were approved on October 7, 1976.

### DISCUSSION (of information and data considered in the development of the guidelines)

#### 1.0 Background (data up to October 1976)

The literature devoted to the effects of asbestos fibre exposure is extensive and reflects the profoundly disturbing biological impact of the mineral on industrialized societies since the mid forties.

The full potential of the morbidity and the mortality resulting from exposure has not yet been fully demonstrated and may not be fully known.

The significance of findings in experimental animals is not clear and most have not been duplicated in humans. Even in diseases where there is a clear cause and effect association accepted, eg., lung cancer, mesothelioma and asbestosis, the mechanisms involved in cell damage are obscure and the dynamics of fibre migration remain unexplained. The overall impression, however, is clear. Asbestos fibre has the capability of causing a broad spectrum of cellular reaction in humans from benign to fatal effects.<sup>1</sup>

<sup>1</sup> G. Ostigy, Health Hazards of Asbestos-Exposure, Preliminary Report (Montreal: University of Montreal, 1975).





Further studies have shown that occupational exposure involved ingestion of a considerable number of fibres which can subsequently be readily identified by stool analysis. The number found depends upon the intensity of the exposure.<sup>2</sup>

## 2.0 Findings to 1976 - Gastrointestinal Cancers:-- (Esophagus, Stomach, Small bowel, Colon and Rectum)

The first indication that gastrointestinal cancers might be increased in asbestos workers was reported in 1964 among 632 insulation workers with more than 20 years of exposure. The increase appeared to be about 3 times normal. This study was subsequently expanded eventually comprising nearly 17,800 individuals. (Appendix 3)

Over the next 10 years, other investigators had also reported a suggestive association between asbestos exposure and gastrointestinal cancers. These studies were all evaluated by Dr. A.C. Ritchie and Dr. A.B. Miller in their reports. (Appendices 3, 4 & 5) Some of these studies were not relevant as statistical exercises, since they did not involve properly structured epidemiological populations.

Evidence accumulated on insulation workers in Belfast between 1940 and 1966 suggested at least a twofold increase of GI cancer among them. (Appendix 5) Other studies examined by Dr. Ritchie and subsequently by Dr. Miller appeared to fulfill the criteria for valid epidemiological surveys. However, the time intervals involved were not yet great enough to derive any conclusions.

## 3.0 Site of the Cancer

Dr. Ritchie has indicated that there is a stronger association suggested between colonic cancer and asbestos exposure than between gastric cancer and exposure. While other studies tend to sustain this observation, there is inadequate information available in these other works to clearly indicate the exact site of the cancer along the gastrointestinal tract.

It is Dr. Miller's opinion that since we are dealing with a statistically rare event, the GI tract should be approached from a "unitary" point of view and the assumption made that there would be increases in all types of cancers regardless of their site. The data are not conclusive enough to establish a prime risk area in the gastrointestinal tract--that is, from the upper end of the esophagus down to and including the rectum.

Dr. Miller concludes that it is likely the data currently available are as good as any which may become available in the future. In other words, there is an almost threefold increase in the incidence of gastrointestinal cancer, and future evidence will not likely alter this finding.

<sup>2</sup> Cummingham et al, "Asbestos Ingestion - Fibre Count in Stool", Journal of Toxicol & Environmental Health, Volume I, 1976.





#### 4.0 Summary of the Observations (Appendices 3, 4 & 5)

4.1 There is agreement by the authors of the two studies commissioned by the Board, that a valid epidemiological cause/effect relationship between asbestos exposure and gastrointestinal cancer has been established.

4.2 This statistical increase in the incidence of GI cancer is in the order of two to three times the normal expected.

4.3 It is clear that exposure to asbestos must be "occupational" before a causal relationship can be established.

4.4 The studies do not show any increase in the incident of GI cancer unless the latency period is 20 years or more--that is the time between first exposure and the diagnosis of the cancer.

4.5 The mode of entry of the asbestos fibre which produces these cancers remains unclear. It may be either from ingestion or inhalation.

4.6 There is no well defined dose/response or dust index relationship between exposure and the incidence of GI cancer.

4.7 The conclusions strongly suggest that the gastrointestinal tract, as previously defined, be considered as a unit and that cancer location not be considered a factor in allowance.

4.8 Smoking is not a factor to be considered in assessing GI cancer.

#### 5.0 Definition of Occupational Exposure

5.1 Direct.

5.2 Indirect, Incidental or Proximity.

5.3 Duration.

##### 5.1.1 Direct Exposure:

This includes any job connected with regular production, processing, handling, and the use of asbestos containing materials.  
For example:

##### 5.1.1.1 Asbestos Mining and Milling:

Included in this category would be miners, sorters, asbestos haulers, screeners, crusher operators, mill operators, et cetera.

##### 5.1.1.2 Asbestos-Cement Production & Use:

Most of the asbestos mined in the world today is used in the manufacture of a variety of asbestos cement and plaster



products. Production, processing and commercial use may result in occupational exposure to certain employees, eg., mixers, sprayers, insulators, lagers, shipyard workers, plasterers, masons and engineers.

#### 5.1.1.3 Asbestos-Manufacture of Textiles:

Asbestos alone or in combination is employed extensively in the manufacture of threads, yarns, cloth, sheets, clutch facings, gaskets, et cetera. This would include such occupations as spinners, carters, and weavers who prepare the raw fibre.

#### 5.1.1.4 Asbestos-Miscellaneous:

This would include, for example, jobs involving the use of tile, seals, packing materials, and filters.

#### 5.2.1 Indirect, Incidental or Proximity Exposure:

The level of this exposure is often difficult to evaluate. Such an employee would be one who is permanently or temporarily in or near operations where asbestos and asbestos products are being produced or handled by others, eg., repairmen, maintenance men, engineers, mechanics, laboratory technicians, construction workers, shipyard workers, plumbers, carpenters, sheet metal workers.

Since approximately two-thirds of all asbestos produced is used in construction products, the potential for proximity exposure is high.<sup>3</sup> Therefore, it should be considered that occupational exposure means that type of exposure in a job which is recognized as one primarily involved in asbestos handling. It should not include proximity or incidental exposure unless shown that such exposure, even if intermittent, is of such intensity as to have a known relationship to disease, eg., sheet metal workers, iron workers, plumbers, who have had permanent or semi-permanent employment in shipyards.

#### 5.3.1 Duration of Exposure:

No information is available from the consultants' reports with respect to a specific duration of exposure occurring within the 20 year latency period.

It is impossible to evaluate the weight of heavy early, but discontinued exposure, as compared to light but continuous exposure over the time interval mentioned. While it is recognized that heavy, short duration exposures of a few months may lead to lung cancer or mesothelioma, it would appear that the same probably does not apply to the GI tract. Risk of asbestosis or lung cancer probably depends on the duration and the quantity of fibre retention in tissue. This concept clearly applicable for the lung, cannot necessarily be applied to the GI tract. Therefore, occupational exposure should include some element of continuity or repetitiveness over a period of years.

<sup>3</sup> Air Resources Branch, Government of Ontario, Asbestos as a Hazardous Contaminant II, Ministry of the Environment, January, 1975.



GASTROINTESTINAL CANCER - ASBESTOS WORKERS  
GUIDELINES FOR ADJUDICATION

1. That gastrointestinal cancer in asbestos workers be accepted as an industrial disease under Section 118 and 1(1)(L) of the Act as peculiar to and characteristic of a process, trade or occupation involving exposure to asbestos.

2. That based on medical studies, gastrointestinal cancer (esophagus, stomach, small bowel, colon and rectum) be favourably considered when the following circumstances apply:

2.1 There is a clear and adequate history of occupational exposure to asbestos dust, and while such occupational exposure cannot be quantitatively described, it should be of a continuous and repetitive nature and should represent or be a manifestation of the major component of the occupational activity.

2.2 There is a minimal interval of 20 years between first exposure to asbestos and the diagnosis of the gastrointestinal cancer.

2.3 That all primary cancers associated with the esophagus, stomach, small bowel, colon and rectum be included in the classification of gastrointestinal cancers.

2.4 That no distinction be given as to the site of the cancer in assessing the merit of a claim.

2.5 Claims which do not meet the guidelines in 2.1, 2.2, 2.3, 2.4, should be individually judged on their own merit having regard to the nature of the occupation, the extent of the exposure and other factors peculiar to the individual case. Consideration will be given where it seems evident that the gastrointestinal cancer resulted from occupational exposure to asbestos. The benefit of reasonable doubt applies.

APPROVED BY THE BOARD

October 7th, 1976





LARYNGEAL CANCER RELATED TO  
EXPOSURE TO ASBESTOS & EXPOSURE TO NICKEL

INTRODUCTION

1. Dr. A.C. Ritchie's Studies

In 1974, Dr. A.C. Ritchie, Pathologist, Toronto General Hospital, was requested by the Board to review World Literature relative to asbestos exposure and occupational diseases with specific reference to lung cancer. Dr. Ritchie submitted his initial report (Appendix 2) in 1975, indicating (item 85) that several reports in the literature had suggested a possible relationship between asbestos fibre dust and laryngeal cancer.

In his supplemental report to the Board in April 1976 (Appendix 3), he again mentioned that according to several articles in the literature, exposure to asbestos appeared to increase the risk of developing carcinoma of the larynx. However, he cautioned that this association remained to be proven.

In his final report to the Board, July 27, 1976 (Appendix 4), he again referred to the assumption that exposure to asbestos fibre dust may increase the risk of carcinoma of the larynx, but once more stated that this relationship requires further proof.

2. Dr. A.B. Miller's Studies

The Board contacted Dr. Anthony Miller, Director, Epidemiology Unit, National Cancer Institute of Canada, on October 18, 1976, and discussed the matter of laryngeal carcinoma and its relationship to asbestos fibre dust. Subsequently, a series of meetings took place during which Dr. Miller agreed to develop a case control study on behalf of the Board to evaluate this problem. During the course of discussions, it was suggested to Dr. Miller that he evaluate any effects from nickel aerosol as well as asbestos fibre dust, primarily to develop another specific area of enquiry and also to settle the question which had been raised from time to time relative to the relationship between laryngeal cancer and nickel aerosols. Dr. Miller agreed and the study commenced in February of 1977.

The actual case interviews commenced in May 1977, after the appropriate questionnaire had been developed and the interviewers trained, but subsequently, Dr. Miller informed the Board that in his opinion, the study could not be completed on the basis of the data assembled, and that for this reason, he had made arrangements for further funding in order to continue the study into the second year.

At the Board's request, Dr. Miller developed an interim report based on 62 matched pairs of laryngeal cancer cases (up to January 1978) and presented it to the Board's senior medical staff on April 11, 1978 (Appendix 6).



At that time, Dr. Miller stressed that the study was incomplete, but that it would continue for at least one further year, and that he was seeking approximately 200 cases in matched pairs. However, he informed the Board that the initial study had disclosed a most interesting finding which he considered to be statistically significant, and which had not been reported in the literature before, namely, that there appears to be a synergistic, or additive effect, to a person exposed to both asbestos dust and nickel aerosol.

Dr. Miller also indicated that a relationship appears to exist between laryngeal carcinoma and exposure to asbestos fibre dust, as well as laryngeal carcinoma and exposure to nickel aerosols, and that in his opinion, while independently these relationships must be considered borderline in significance, considering the three cases where there had been combined exposure, his study to date in all probability has established a relationship.

In the letter that accompanied his report of April 1978, Dr. Miller stated that:

"In my opinion, there is sufficient evidence to relate cancer of the larynx as an occupationally related disease, and that in all probability, both exposure to asbestos and exposure to nickel are causal factors."

## DISCUSSION (of information & data available to April 1978)

### 1.0 Background

In each of several industrial processes involving an increase in the incidence or the risk of respiratory cancers, the Board has developed criteria or guidelines for the acceptance of such cancers based on clear evidence of mortality increase in well defined occupations, based on prospective studies of selected groups instituted in Ontario or elsewhere. While the epidemiological approach has varied somewhat according to the prime data available, in the majority of cases, abnormal occurrence within specific industrial exposures has been demonstrated. By comparing observed mortality with that expected in exposure duration sub-groups, estimate of risk, latent period and exposure duration necessary to establish acceptance criteria, became available (nickel refining, precious metal refining, coal gasification, coke-oven exposure).

Those studies involving nickel refining revealed several increased-risk occupations inside the industry, not associated with any one identifiable carcinogen, but linked to two particular respiratory malignancies.

A prospective study done in the case of Consumer's Gas Company clearly showed an increase in lung cancer in workers who worked on the horizontal retorts, but not on the vertical retorts, i.e., revealed a specific risk area within the one industry. Similar studies done on the steel industry in the U.S.A. by Lloyd revealed specific risk occupations within that industry, mainly restricted to the coke-ovens.

Similarly in the case of the Doloro Mining and Smelting Company, specific risk occupations were identified through prospective studies, within the precious metal refining process.





In the same manner, prospective studies in many countries have clearly demonstrated increase in respiratory cancer in the manufacture of asbestos products and in the milling and mining of asbestos. However, a differential effect was noted between some occupations, but the increased risk was considered to be clearly applicable in varying degree to all occupations using asbestos as a principal product.

Relative risk differentiation has also been demonstrated (but not as clearly) in uranium mining<sup>1</sup>, since the carcinogen is ubiquitous underground and all occupations are variously affected in much the same way as in the manufacturing and fabricating of asbestos products.

Two of the malignancies covered in the various guidelines (sinus and mesothelioma) are so rare as to preclude the necessity of identifying specific risk occupations.

According to Dr. Sutherland<sup>2</sup>, the chief criteria for accepting lung cancer as an occupational disease are:

- (1) Statistical evidence of increased frequency of occurrence in a particular occupational population group.
- (2) Demonstrating increased frequency of the disease with increasing duration of exposure.

## 2.0 Laryngeal Cancer - Nickel Refining

Virtually none of the above factors are operative in the case of laryngeal cancer. Consider the following:

- a) It is not so rare, as in the case of sinus cancer or mesothelioma, that it can be considered occupational in all cases, i.e., no automatic presumption of cause and effect.
- b) No epidemiological increase in laryngeal cancer has been demonstrated in the various nickel refining processes in Europe<sup>3</sup> & <sup>4</sup> or North America with one exception - the Pedersen study in Norway. (Refer to point 8.0)

<sup>1</sup> Muller et al, "Causes of Death in Ontario Uranium Miners," International Symposium on Radiation Protection (France: September 1974).

<sup>2</sup> Dr. R.B. Sutherland, "Recognition of Lung Cancer as an Occupational Disease," Canadian Public Health Association (Regina: Convention, May 1961).

<sup>3</sup> R. Doll et al, "Cancer of the Lung and Nasal Sinuses in Nickel Workers," British Journal of Cancer, Volume XXIV, 1970.

<sup>4</sup> A.V. Sakmyn et al, "Some Statistical Materials in the Production of Nickel on an Ore Oxide Base," Gigiena Truda (Professor Zabol, November 1970).





- c) No supportive evidence exists of increase in either the mortality rate or case incidence of laryngeal cancer in the Port Colborne or Copper Cliff work force between 1930 and 1974.<sup>5,6 & 7</sup>
- d) No epidemiological studies available of individuals who have been exposed to nickel compounds in non-refining processes, i.e., nickel welders, nickel platers, nickel grinders, or in chemical workers using nickel catalysts, show evidence of increase risk.<sup>8</sup>
- e) No common occupational exposures within the nickel refining process in the Ontario cases are apparent.

## 2.1 Ontario Vital Statistics - Laryngeal Cancer

In 1965, 199 new cases of laryngeal cancer in men were identified. In the same year, 60 deaths were recorded from laryngeal cancer.<sup>5</sup>

In the case of women, there were 20 cases identified and 7 deaths.

The ratios (living - dead cases) are consistent (i.e., 3.3/1) and reflect the known five year survival from laryngeal cancer at about 60% to 70%.

The figures from the 1977 publication of the Ontario Cancer Treatment and Research Foundation<sup>9</sup>, show 318 new cases of laryngeal cancer and 125 deaths from laryngeal cancer in 1976 (i.e., 2.6/1). It is clear, therefore, that in assessing incidence of laryngeal cancer in industry, one has to assume that there are between 2.5 to 3.5 cases of laryngeal cancer present for every death reported due to this disease.

## 2.2 Surveillance of Nickel Refining in Ontario

Since 1958 (up to 1974), occupational histories were obtained by the Occupational Health Branch, Ontario Ministry of Health in all cases of cancer death from the Port Colborne and Copper Cliff work force. (Search commenced in 1948.) All cancers were assessed including laryngeal.

<sup>5</sup> Ontario Vital Statistics - 1965.

<sup>6</sup> "Nickel and Its Inorganic Compounds," A supplementary submission to National Institute of Occupational Safety and Health (United States: Inco - October 1976).

<sup>7</sup> Dr. R.B. Sutherland, "Personal Communications," April 18, 1978.

<sup>8</sup> F.W. Sunderman, "A Review of the Metabolism and Toxicology of Nickel," International Symposium on Clinical Chemistry and Toxicology of Metals, March 4, 1977.

<sup>9</sup> "Cancer in Ontario," The Ontario Cancer Treatment and Research Foundation, 1977.



### 2.3 The Inco Port Colborne Experience

In 1974, according to a letter from Dr. R.B. Sutherland<sup>10</sup>, out of seven known cases of laryngeal cancer, four were living and three were dead. This represented the total number of laryngeal cancers occurring in the Port Colborne work force between 1930 and 1974. The work force varied between 800 to 1,200 during the period under surveillance.

In respect to the expected number of laryngeal cancers appearing in the work force at Port Colborne, in a publication by NIOSH<sup>6</sup>, it was calculated that 2.1 deaths were to be expected in the Port Colborne work force between 1930 and 1974. If the ratio of living to dead cases is 3.3 to 1, then the total number of cases deduced from this mortality figure would be seven - this is almost precisely the number of cases which were known to exist from the Port Colborne work force in 1974. This should be compared with the Copper Cliff experience.

### 2.4 The Copper Cliff Experience

Between 1930 and 1974, 12 deaths from laryngeal cancer are known to have occurred<sup>7</sup> in the Copper Cliff work force (12,000 to 15,000 average), i.e., 2 to 3 deaths/100,000 man years. This figure contrasts with Ontario Mortality Rate (1971 to 1975) from laryngeal cancer, all ages at 2.2/100,000 population (male)<sup>9</sup>. For the age group (30 to 65), a comparable figure would be approximately 3 to 7/100,000 population (male) which is closer to Port Colborne experience, i.e., 3 to 4/48,000 man years.

### 2.5 Case Distribution

The distribution of the Port Colborne cases in respect to occupation gives no hint of any common exposure, and suggests no more than that expected from the employment ratio in the jobs involved. Only two had minor exposure to hazard employment. Three individuals had over twenty years each in the electrolytic and anode department, while four others had varying numbers of years as pipe fitters, welders, precious metal workers, labourers, etc.

In summary, the number of laryngeal cancer cases from the Port Colborne work force are not more than expected from the period 1930 to 1978. Furthermore, there have been no reports of increase of laryngeal cancer in world-wide nickel operations other than in a Norwegian study by Pedersen. This is examined.

<sup>6</sup> Idem.

<sup>7</sup> Idem.

<sup>9</sup> Idem.

<sup>10</sup> Dr. R.B. Sutherland, "Personal Communication, Laryngeal Cancer - Port Colborne Refinery," February 9, 1974.





## 2.6 The Pedersen Study<sup>11</sup>

Pedersen stated in his report that his figures do confirm the existence of a very substantial health hazard amongst the men involved in roasting, smelting, and electrolysis.

However, Dr. R.B. Sutherland, in his analysis of the Pedersen Study<sup>7</sup>, ascertained that there was not a single case of laryngeal cancer which had appeared from the electrolytic process. Four cases had appeared in the roasting and the smelting process, and one in a miscellaneous process.

The problem here is that Pedersen combines mortality rates and cases, and the case incidence is not the same as mortality rate for laryngeal cancer, as it is in the case of say lung cancer where the case incidence and the mortality rate may be considered as equal. His analysis is not clear.

In the five cases reported (between 1936 and 1960), the duration of employment averaged nine years with a range between 4 and 16 years. The average latent period was 15.6 years, with a range of 8 to 31 years.

This contrasts with the experience at Port Colborne where most of the exposures were over twenty years spread over the following occupational exposures: precious metal refining, electrolytic refining, anode furnace, welding, shear labour, mechanical department, etc. The breakdown is noted in Table I, on page 44.

## 2.7 Miller Study - Interim Report<sup>12</sup> (Appendix 6)

Dr. Anthony B. Miller, Director, Epidemiology Unit, National Cancer Institute of Canada, undertook an epidemiological study of cancer of the larynx after discussions with the Workmen's Compensation Board, Dr. A.B. Ritchie of the Toronto General Hospital and Dr. Douglas Bryce, Professor of Otolaryngology at the University of Toronto. This study was authorized in February of 1977 to obtain information not only on asbestos exposure but on exposure to nickel.

On April 11, 1978, Dr. Miller reported in person to the Board and delivered his report. In a covering letter, he stated that while a final conclusion on the etiologically relevant role of either asbestos or nickel is not yet attainable, ... "we will probably conclude that both asbestos and nickel exposure under appropriately defined circumstances are causally associated with cancer of the larynx." Based upon the studies done so far, Dr. Miller concluded: "In my opinion there is sufficient evidence to relate cancer of the larynx as an occupationally related disease and that, in all probability, both exposure to asbestos and exposure to nickel are causal factors."

<sup>7</sup> Idem.

<sup>11</sup> Pedersen et al, "Cancer of Respiratory Organs Among Workers at a Nickel Refinery in Norway," International Journal of Cancer - Volume XII, 1973.

<sup>12</sup> Dr. A.B. Miller, "Asbestos and Carcinoma of the Larynx" - Interim Report, National Cancer Institute of Canada, April 7, 1978.





In his conclusion, Dr. Miller reported that "exposures of the type which may well be relevant to the aetiology of laryngeal cancer are not identified through routine occupational histories. Special questioning is required in order to elicit information on such exposure."

No information is available from these data with respect to latent periods, intensity of exposure, the likely carcinogen or the particular industrial process or occupation wherein the hazard is encountered. (Table II on p. 45)

Similarly, no common occupational exposure is apparent within the Port Colborne data. (Table I on page 44)

Exposures apparently common to both groups are restricted in two cases to work in precious metal refining (21 years and 5 years) and in three cases to the mining of nickel ore (23 years, 14 years, 41 years).

## 2.8 Laryngeal Cancer in Industry (excluding Nickel Refining)

Sunderman has examined nickel carcinogenesis in great detail<sup>8</sup>, 13 & 14, and noted that the only epidemiological studies in respect to respiratory cancers in occupational nickel exposure relate to the refining and the smelting of nickel.

He has shown that several of the constituents of ordinary nickel rafter dust are carcinogenic in animal experiments, notably nickel sulphide ( $\text{Ni}_3\text{S}_2$ ).

In 1962, a sample of refinery rafter dust was analyzed from an Ontario Nickel Refinery and the following constituents were found:  $\text{Ni}_3\text{S}_2$ ,  $\text{NiONi}_2\text{O}_3$ ,  $\text{NiSO}_4\text{H}_2\text{O}$ ,  $\text{CoS}$ ,  $\text{CuS}$ ,  $\text{CuO}$ ,  $\text{Cu}_2\text{S}$ ,  $\text{CoO}$ ,  $\text{FeS}$ ,  $\text{FeO}$ , and  $\text{Fe}_2\text{O}_3$ .

He considers the insoluble dusts, nickel sulphides, nickel oxides, and the soluble aerosols of nickel sulphate, nitrate, or chloride, are the most harmful.

In 1977, he recorded that there were only three previous case reports of work-related cancers in the respiratory tract in workers outside of nickel refining, i.e., exposed to inhalation of nickel and nickel plating, and grinding.

He personally records a fourth case involving a nickel stripping process in which small nickel plated items were stripped of their own nickel plating to expose the copper base. The author felt that the nasal cancer appearing in this worker was occupational in origin and caused by nickel.

<sup>8</sup> Idem.

<sup>13</sup> F.W. Sunderman, "Carcinogenic Effects of Metals," Symposium Biological and Pharmacological Effects of Metal Contaminants (Chicago: April 8, 1971).

<sup>14</sup> F.W. Sunderman, "The Current Status of Nickel Carcinogenesis," Annals of Clinical Laboratory Science, Volume III, 1973.



Sunderman goes into great detail in respect to the cancers induced in experimental animals by the administration of various nickel compounds, but he records not a single case of laryngeal cancer in non-refinery workers, although he does list at least four known sinus or lung cancers in this group.

## 2.9 Summary

There is no apparent increase in either the mortality rate or the case incidence of laryngeal cancer in the Inco nickel refining operations.

There is no common occupational exposure demonstrated in those cases of laryngeal cancer known to the Board and associated with known exposure. We have no knowledge of the occupational exposure nor are we able to identify an estimated two to three dozen cases of laryngeal cancer from the Copper Cliff work force between 1930 and 1974.

There is no common occupation revealed from the initial results of the case study questionnaire in those who have some nickel exposure.

The only two common exposures noted between these two main groups are that of underground mining and precious metal refining exposure.

Laryngeal cancer has not shown to be increased by epidemiological studies of the Welsh, Canadian or Russian nickel refining processes. The only report of an increase in this malignancy is from a Norwegian refinery work force.

The bulk of exposures in the Port Colborne laryngeal cancer group are twenty or more years as opposed to the average in the Norwegian group of nine years. The range of exposures and this latter average more closely approximate those of the Miller case control study group. The Miller cases, however, exhibit no common occupational exposure. The significance of this approximate similarity, therefore, is very doubtful.

In the three case control dual exposures of asbestos and nickel in the Miller study it is difficult to discern a possible synergism, since in two of these cases the asbestos exposure alone, if confirmed, might be interpreted as being significant and in only one does there appear to be a possible significant nickel exposure, although even this is questionable.

No such synergism is reported in the world literature and Sunderman never speculates on it in his in-depth examination of the biological effects of nickel, despite the fact that nickel is often found as an impurity in chrysotile and as a minor substitute for magnesium in the crystal lattice<sup>15</sup>.

However, some researchers have attributed the carcinogenic potential of asbestos to some of the trace metals found in the crystal lattice<sup>16</sup>.

<sup>15</sup> S. Speil et al, "Asbestos Minerals in Modern Technology," Environment Research.

<sup>16</sup> "Asbestos as a Hazardous Contaminant II," Air Resources Branch, Ministry of the Environment, January 1975.





Eight trace metals are known including nickel. Chrysotile was found to have greater amounts of nickel and chromium, which may not always be incorporated structurally in the fibre, but be associated with other impurities. Since magnesium is leached out in vivo, nickel might be expected to react in the same way.

## 2.10 Laryngeal Cancer - Nickel Exposure and Compensation

The only evidence of any substance which relates laryngeal cancer to nickel exposure is Pedersen's study. The Miller study lends weight only if the three dual exposure cases are included. Assuming that there is some degree of occupational cause and effect in each of these three cases, it seems much more likely that it is due to asbestos alone, and there is no need to seek an explanation elsewhere or to postulate some sort of synergism between nickel and asbestos.

The case for an association seems much stronger for asbestos than for nickel. There is only one incident where nickel might be of significance in the dual exposures and that is linked with thirteen years of asbestos lagging which in itself may be significant.

The Pedersen study results also highlights a fact about nickel refining-- that it is parochial in its effects. There has been no increase in respiratory cancers in the Coniston smelter of Falconbridge in Sudbury and one, therefore, cannot extrapolate a finding from one geographical location to another. There is no more reason to put any more weight on the validity of the Norwegian studies than on the case incidence or mortality rates known in the Inco working population.

## 2.11 A Formula for Compensation

Dr. Miller has indicated that in his opinion, and based upon his incomplete study that the Board has no choice but to accept laryngeal cancer in nickel refining. The present guidelines for lung or sinus cancer which have been designed to incorporate malignancy rates hugely increased over normal, i.e., 50 to 100 times for sinus cancer and about 7 to 10 times for lung cancer cannot be used for laryngeal cancer. If the Board takes all known cases of laryngeal cancer at Inco, and those with significant nickel exposure in the Pedersen study, the shortest exposure would seem to be around 7 years (in non-hazard operations) with the great majority over 20 years. The average exposure for the Pedersen cases is 9 years. However, because there is a clear clustering of cases in the Pedersen study to refinery process, not duplicated in the Ontario experience, there would be no justification to base guidelines on these findings.

2.11.1 Based upon the Board's analysis, the exposure period of 20 years is appropriate except in previously identified hazard areas for lung and sinus cancer where a minimum period of 5 years would be reasonable.

2.11.2 The latency period (inception) based on analysis, should be 20 years or more.





### 3.0 Asbestos and Laryngeal Cancer

#### 3.1 The Selikoff Studies

Selikoff reported his findings regarding asbestos exposure in 17,800 insulation workers<sup>17</sup>. These studies have never been officially published, although on October 24, 1977, he reported an analysis up to January 1, 1977. There was an increase in laryngeal cancer represented by 11 observed cases against 4.84 expected. There were some uncertain elements in this analysis since the lack of a smoking history exposure suggested a confounding element in the picture which would have to be resolved.

#### 3.2 The Miller Study - Preliminary Report<sup>12</sup>

This report suggests that there is nothing in the results of this study which invalidates the findings of Selikoff and there are in fact now, in Miller's judgement, no confounding factors which will explain this increase such as alcohol or smoking.

In general, there were no clues as to the severity of exposure which might be required, or an appropriate latent period.

Table III on page 46 presents a listing of exposures revealed by the questionnaire in which there might be significant exposure to asbestos fibre.

The distribution gives no clues as to the quality or the intensity of the exposure and the duration varies widely. The latent periods are mostly 25 years or more, similar to the Stell and McGill results<sup>18</sup> with the average duration of exposure probably a little less.

It is still difficult to discern a synergistic effect by including the three dual cases in this Table (as was the case in Table II).

#### 3.3 The Stell & McGill Study<sup>18</sup>

This was a matched case control study which was first published in 1973. The study was expanded by 1975 to a total of 119 cases of laryngeal carcinoma with 119 controls. Three types of occupational exposures were recognized:

- a) lagging heater equipment
- b) scaling of boilers
- c) unloading raw asbestos.

<sup>12</sup> Idem.

<sup>17</sup> I.J. Selikoff et al, "Laryngeal & Bucco - Laryngeal Cancers in Asbestos Workers," 3rd International Symposium on Detection & Prevention of Cancer (New York: 1976).

<sup>18</sup> Stell & McGill, "Asbestos & Laryngeal Carcinoma," Lancet, August 1973.



There were no essential differences in smoking habits, but there were a greater number of carcinomas in the smoking group.

The average age of onset of the carcinoma was 10 years or so less in patients exposed to asbestos than in those with no exposure, i.e., 42% were between 51 and 60 years at onset in the exposed group as opposed to 48% in the 71 to 80 year old group in the non-exposed group.

The latent period averaged 30 years and the average duration of exposure was 27 years.

The authors suggested that the weakness of the association related to the methodology, i.e., a retrospective study, but contended that a prospective study of the problem will be very difficult because of the relative rarity of this cancer and would be difficult to collect a static population of asbestos workers "large enough to produce sufficient cases of laryngeal cancer for a prospective survey."<sup>18</sup> They related that only one of their laryngeal carcinoma cases had asbestosis, and surmised that those individuals who might have developed laryngeal cancer in the past on account of heavy exposure did not do so because death from asbestosis occurred first and that this class of patient represented the results of lesser exposure wherein the individual survives long enough to develop cancer - hence the long latent period discerned in their survey.

Smoking habits were not confounding and while smoking may be a co-factor in the development of laryngeal carcinoma, "it is unlikely to be important since the incidence of laryngeal cancer in the 20th century has declined slightly in the face of an enormous increase in tobacco consumption."<sup>18</sup>

Dr. M. L. Newhouse<sup>19</sup> reported briefly on the mortality experience from laryngeal carcinoma at an asbestos textile and cement factory. Among a cohort of over 4,000 individuals followed since 1933, there were two deaths from laryngeal cancer, one with 13 months exposure starting in 1939 (death 1963) and the other an employee for one month in 1949 (death 1962). High intensity dust exposure was identified in both cases.

### 3.4 Laryngeal Cancer, Asbestos Exposure and Compensation

The case for linking laryngeal cancer and asbestos is probably as strong as that linking GI cancer and asbestos, given the latest data from the Miller report and the previous Selikoff findings. While Dr. Miller comments on the duality of exposure and possible synergism, it is not necessary to postulate such a relationship in the three cases concerned since in each asbestos might be considered significant in isolation.

In the Stell and McGill studies, the average duration of exposure was 27 years with a latent period of 30 years. Since only one of their cases had

<sup>18</sup> Idem.

<sup>19</sup> M.L. Newhouse, "Asbestos and Laryngeal Carcinoma," Lancet, September 5, 1973.





asbestosis, it could be assumed that the intensity of the exposure generally was mild to moderate only, and was long in duration. The Miller case control results are not dissimilar with most of the latent periods more than thirty years and none less than sixteen years.

Since Stell and McGill found that the average age of onset was 10 years less in the exposed as opposed to the non-exposed, this could be taken into consideration.

### 3.5 The Case for Synergism - Asbestos and Nickel

It is impossible to discern or interpret synergism from the data at hand. Its postulation rests on three cases and only one of these is impressive. The fact that it has not been reported in the world literature, either in experiment or in epidemiological survey, undermines the assumption. Nickel is known to be an impurity in the chrysotile crystal and sometimes replaces magnesium, but Sunderman makes no comment on it.

Based upon all data, including the Miller study, up to April 1978, and considering that in the three dual cases the latent periods are 16, 35 and 47 years, it would be reasonable to accept the same latent period for duality as in single exposures. However, it has been recommended by Dr. Miller that consideration be developed to reflect a possible synergistic effect. To this end, it is recommended that recognition of duality can be accomplished by an adjusted inception period to 15 years and by a halving of the accumulated exposure intervals.

### CONCLUSION (as at May 1978)

Despite the fact that the studies had not been completed, it had become evident that Dr. Miller had established a probability of a relationship, not only between laryngeal cancer and exposure to asbestos fibre dust, but also between other factors such as laryngeal cancer and nickel aerosol exposure, and the totally new finding of the apparent synergistic or additive effect on a person who is exposed to both hazards.

In view of Dr. Miller's findings and the other considerations, it was concluded that the Board should accept this relationship and proceed with the development of interim guidelines for the Medical and Claims adjudication of these types of case.

In addition, Dr. Miller was able to supply the Board with one very important factor, namely that of latency (or inception) period from time of first exposure to onset of the cancer. This factor was utilized in the development of the guidelines. Furthermore, discussion with Dr. Miller allowed the Board to utilize the exposure and intensity factors, previously proven and adapted into some of the existing guidelines relating to lung cancer, gastrointestinal cancer, and mesothelioma. Finally, evaluation of other studies (relating to nickel aerosol) carried out by the Board's Consultant in Chest Diseases, allowed the Board to develop similar guidelines relative to nickel aerosol exposure.

On this basis, the guidelines for the adjudication of claims for laryngeal cancer in industry related to asbestos exposure and nickel exposure were approved by the Board on May 4, 1978.





GUIDELINES FOR ADJUDICATION OF CLAIMS FOR LARYNGEAL  
CANCER IN INDUSTRY RELATED TO ASBESTOS EXPOSURE AND  
NICKEL EXPOSURE

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RECOMMENDATION

1. Laryngeal cancer in workers occupationally exposed to asbestos fibre and/or to nickel aerosol in specific industrial processes be accepted as an industrial disease under Section 118 and Section 1(1)(L) of the Act as peculiar to and characteristic of such processes.
2. Based on medical studies, claims for laryngeal cancer be favourably considered under the following circumstances:
  - 2.1 Any industrial process in the nickel industry which produces nickel in aerosol dispersion whether in combined or elemental form. This may include the following:

- Roasting	- Welding
- Smelting	- Electroplating
- Refining	
  - 2.2 Any occupation in which there is a clear and adequate history of occupational exposure to asbestos dust, and while such occupational exposure cannot be quantitatively described, it should be of continuous and repetitive nature and should represent or be a manifestation of the major component of the occupational activity.
3. Duration of Exposure
  - 3.1 Nickel - an accumulative minimum of 15 years exposure to nickel aerosols as defined in 2.1.
  - 3.2 Asbestos - an accumulative minimum of 10 years proven exposure as defined under 2.2.
  - 3.3 Nickel and Asbestos - an accumulative minimum of 7.5 years nickel as well as 5 years asbestos exposures in the case of dual exposure.
4. Inception Period
  - 4.1 Nickel - this shall be a minimum of 20 years from the commencement of the first hazardous exposure.
  - 4.2 Asbestos - this shall be a minimum of 20 years from the commencement of the first hazardous exposure.
  - 4.3 Nickel and Asbestos - this shall be a minimum of 15 years from the commencement of the first hazardous exposure.
5. Claims which do not meet the guidelines should be individually judged on their own merit having regard to the intensity of exposure and other factors peculiar to the individual case. The benefit of reasonable doubt applies.

"Approved by the Board"

May 4, 1978.



TABLE I

EXPOSURE RECORD FOR LARYNGEAL CARCINOMAS - PORT COLBORNE 1930-1978

CASE	ANODE	ELECTROLYTIC	PRECIOUS METAL	MECHANICAL	REMARKS
	Yrs	Yrs	Yrs	Yrs	
1	23				crane operator
2		21			tube filterman process labour 3 years
3			21		operator
4		24			various
5				22	pipe fitter shop labour
6				23	welder yard
7	3	0.5		4	brief exposures to calcining & P.M.
8	2 months	6		2	22 months calcining & leaching



TABLE II

NICKEL EXPOSURE DURATION FOR CASES ANALYZED IN MILLER REPORT

CASE	MINING Ni	SMEETING REFINING Ni	TRANSPORT Ni	STEEL MAKING	REMARKS
	Yrs	Yrs	Yrs	Yrs	
1	14				Inco
2	2	3	7 hauling NiO		13 yrs. (1) asbestos lagger
3			7		train conductor nickel ore
4				36	asbestos (2) insul., 5 yrs.
5		8?			8 yrs. Union Carbide
6					47 years. Ni steel sheets 47 years asbestos (3) insul.
7		5			P.M. dept. Inco labour
8				10	blast furnace
*	41	SUDBURY BASIN			W.C.B. case not from study
*		6 Sintering	COPPER CLIFF		W.C.B. case





TABLE III

ASBESTOS EXPOSURE DURATION FOR CASES ANALYZED IN MILLER REPORT

CASE	CONSTN.	LAGGING	CEMENT PIPE	TRANSPORT ASBESTOS	INSUL.	DRY WALL	REMARKS
	Yrs.	Yrs.	Yrs.	Yrs.	Yrs.	Yrs.	
1					8		1969-77 spraying
2	48						asbestos sheeting
3			60				plumber asb. paste
4				45			slali & powder
5				25			trucker 1952-77
6	17 18 yr. L						asbestos sheeting 1959-76
7		2 mos. 30 yr. L					1948 cloth & paste insul.
8						12 30yr.L	1945-77 plasterer
9					12 35yr.L		1944-75 insul.wire
10	5 45 yr. L						flooring 1924-29
11			10 50 yr. L				1915-25 brake lin.
12		13 16 yr. L	Nickel oxide transp. 1963-70				home insul. 1960-73
13			Nickel steel making		5 35yr.L		steel furn. insul. 1940-45
14	47		Nickel sheeting 47 years				asbestos sheeting elevators insul.

L = Latency



## PROCEDURAL GUIDELINES FOR CLAIMS ADJUDICATORS

### - ASBESTOS RELATED CANCER CLAIMS -

1. Send Employee's Report of Occupational Disease, Form 6S, to worker; Employer's Report of Occupational Disease, Form 7S, to employer; Doctor's Report of Occupational Disease, Form 8S, to attending doctor.
2. Approved follow-up and jacket mark-up procedures apply.
3. In cases where normal enquiries do not provide sufficient information, arrange for an investigation by directing a memo through the Team Co-ordinator.
4. Refer claim with a memo to Consultant, Chest Disease, through the Team Co-ordinator. Include the following:
  - worker's occupation
  - outline exposure history
  - diagnosis
  - request an opinion of compatibility of diagnosis to exposure history
5. If not compatible, direct memo to Review Branch, through the Team Co-ordinator, with recommendation for denial. (Usually G2(4) or (5) clauses apply)
6. If compatible:

#### Notification

- a detailed letter to worker, copies to the employer and other representatives, if applicable, outlining the allowance and the benefits to be paid

#### Payment

- consider temporary total and temporary partial benefits if treatment is being rendered
- consider pension review if no active treatment is taking place
- in case of death, consider dependancy benefits

#### 7. Charging of Costs

- there is no S.I.E.F. relief for cancer and chest disabilities
- if there is more than one exposure employer, the costs of the claim are charged to the last exposure employer. For example, if the worker had been employed:
  - 5 years exposure with employer "A" (1945 - 1950)
  - 10 years exposure with employer "B" (1950 - 1960)
  - 10 years exposure with employer "C" (1960 - 1970)
  - 9 years exposure with employer "D" (1970 - 1979),



costs of the claim would be charged to employer "D" - the last exposure employer.

## 8. Benefits

Claims submitted for cancers and chest disabilities usually result in the payment of a permanent disability award, but on occasion temporary total and temporary partial difference payments will apply.

### 8.1 Permanent Disability Wage Basis

- if the worker was in exposure employment at the time the diagnosis was made (or an earlier date, if approved by the Board's Consultant, Chest Disease) the earnings obtained are for the year immediately prior to the date of diagnosis
- if the worker is no longer employed in exposure then comparable earnings of those of a worker working for the same employer, doing the same job, are obtained for the year immediately prior to the date of diagnosis

### 8.2 Temporary Total Benefits

- if active treatment is taking place
- same basis is used as for permanent disability, (see 8.1)

### 8.3 Temporary Partial Difference Benefits

- if the worker is requested to change employment and this change is recommended by his doctor and approved by the Board's Consultant, Chest Diseases, entitlement exists for payment if there is a wage loss
- the permanent disability wage basis is compared with the worker's present wage basis
- payment is granted until no difference exists or a pension is awarded

### 8.4 Dependency Benefits

- in case of death, consider whether the cause was due to cancer or chest disability
- memo should be sent to the Consultant, Chest Disease, with a recommendation
- see "Fatal Claims" for awards payable

NOTE: For copies of the report forms, etc., mentioned in these guidelines, please refer to Appendix 7.





STATISTICS ON ASBESTOS RELATED CANCER CLAIMS  
UPDATED TO DECEMBER 31, 1980

ALLOWED CLAIMS

YEAR	<u>TYPE OF CANCER</u>					TOTAL
	GASTROINTESTINAL	LARYNGEAL	LUNG	MESOTHELIOMA	SINUS	
Prior to 1976	-	-	24	12	-	36
1976	1	-	10	5	-	16
1977	3	-	2	4	-	9
1978	3	2	6	2	-	13
1979	-	1	4	12	-	17
1980	-	1	6	13	-	20
	-	-	-	-	-	-
TOTAL	7	4	52	48	-	111

DENIED CLAIMS

YEAR	GASTROINTESTINAL	LARYNGEAL	LUNG	MESOTHELIOMA	SINUS	TOTAL
*	7	9	11	5	-	32

\*The year in which these claims were denied is not readily available.



IDENTIFICATION PROGRAMME FOR ASBESTOS WORKERS SUFFERING FROM  
GASTROINTESTINAL CANCER, LARYNGEAL CANCER, LUNG CANCER, MESOTHELIOMA

On October 8, 1976, the Public Affairs Division (now the Communications Division) issued the following news release:

"In a press conference called today by the Workmen's Compensation Board, Dr. W. J. McCracken, Executive Director of the Rehabilitation Services Division, released the reports of Dr. A. C. Ritchie, Specialist in Pathology with the Department of Pathology, University of Toronto, and Dr. A. B. Miller, Director of the National Cancer Institute Epidemiological Unit of the University of Toronto. The reports examined the possible relationship of gastrointestinal cancer in workers exposed to asbestos.

The findings of Doctors Ritchie and Miller have established a valid epidemiological cause/effect relationship between asbestos exposure and some gastrointestinal cancers. The studies show an increase in the incidence of GI cancer where the latent period is 20 years or more - that is, the time between first exposure and first appearance of the cancer.

Guidelines for adjudicating these types of claims have now been approved by the Board and the claims involved will be immediately reviewed and a decision rendered within a week."

Subsequently, the Board directed that contacts be established with the Unions and employers involved in the production of and insulation with asbestos, to assist the Board in identifying those workers who might have entitlement for gastrointestinal cancer due to occupational exposure to asbestos.

During the latter part of October, 1976, the Board's staff assembled lists of those Ontario employers who, according to the Board's records, had been, or still were, involved in the production or use of asbestos, so that the past and present members of their executive personnel could be approached for more detailed information on the subject, such as:

- the names and addresses of workers who had died or were known to be suffering from asbestos related cancers;
- the names and addresses of trade associations, companies or installers who had been, or still were, handling asbestos products.

Following the initial contact with the above associations, etc., the plans called for a more detailed discussion with the appropriate union officials in order to confirm the names and addresses of the workers already identified and also for compiling lists of names of those workers who were known as having had worked with or could have had exposure to asbestos.



The last phase of the "identify and seek out" approach called for contacting the respective individual workers, or where the worker had died, - their families.

It was considered that this approach would provide an opportunity for:

- workers, suspected to be suffering from asbestos effects, or cancer, to file a claim with the Board, or for encouraging them to discuss their health with the family physician;
- workers, who had been exposed to asbestos, but who were still in good health, to become aware of the entitlement provisions for asbestos-related diseases, and
- the family of any deceased asbestos worker to become informed of the Board's programme and personnel who could be of help to the family when filing a claim.

Following distribution of a memorandum re Industrially Generated Diseases to members of the medical profession (Appendix 8) in October, the implementation of the above plan commenced in November 1976.

The Ontario Federation of Labour was informed that the Board had commenced contacting the major asbestos manufacturers in Ontario, in order to obtain the names of any of their workers who may be suffering from, or had died from lung cancer, mesothelioma or gastrointestinal cancer. In addition, the O.F.L. was requested that it provide the Board with a list of union locals whose members had been employed in asbestos manufacturing and request the locals to communicate to the Board the names and addresses of any asbestos workers who in their opinion may be suffering from, or who had died of, the above types of cancer.

During phase one of the identification programme, 14 companies were identified as having had very hazardous exposure (to asbestos) environments. During phase two, 17 additional companies were added to the "very hazardous exposure environment" list.

In the meantime, the Occupational Health Branch of the Ontario Ministry of Labour had provided the Board with a list of 99 employers whose employees were considered to have had some exposure to asbestos, and these companies were to be investigated next.

By February 1979, the Board had contacted 29 companies and although the identification programme had not uncovered any claims that could be allowed, it had made a number of employers familiar with the allowance criteria for asbestos-related cancers and, in addition, increased the number of claims (22) reported to the Board for former and present asbestos workers. Besides the above-mentioned contacts, the Board also approached the United Association of Journeymen and Apprentices of the Plumbing and Pipefitters Industry of the United States and Canada, Local 46 (in Scarborough, Ontario), which eventually provided the Board with a list of installers who had died from various causes.





In the most recent discussion on July 9, 1980 with this union, the officials of Local 46 pointed out that they were keenly interested in the identification and assistance programmes and will cooperate with the Board and take the time to search their records and to talk to any long-term employees of the union, so that lists of workers who may have died of asbestos-related cancers could be established. It was pointed out to the union that the Board would be mainly interested in those workers who have had or still have a significant amount of asbestos exposure, or in those who had died of cancer leaving a surviving widow or children.

In March 1979, the Board decided that the identification program should also apply to the shipbuilding industry. A visit was arranged to Collingwood Shipyards in an attempt to obtain more information about other shipbuilding companies that had been active during World War 2, and about the use of asbestos in shipbuilding, generally, at that time.

The contacts with Collingwood Shipyards and various officials of the Department of Supply and Services (Canada) were quite helpful in providing more information on the above subjects. For example, it was established that the types of ships that were built during World War 2 involved Corvettes, Algerine Class Minesweepers, Bangor Class Minesweepers, Fairmiles (a somewhat smaller landing craft) and other types of minesweepers, tankers, cargo ships, lakers and trawlers. The three largest shipbuilding companies in the Toronto area alone each had had up to five docks in production, so that it was not unusual for up to fifteen ships to be launched on any one occasion.

One of the persons contacted, who had in fact served on Corvettes during the war, remembered quite well that asbestos along with a cork mixture was sprayed on the underside of ship decks.

According to the information received from Supply and Services, Marine and Industrial Production, Service Centre, Technical Services Division, in Ottawa, one of the main concerns in shipbuilding was, as it still is, the prevention of fires on board ship. Therefore, areas such as passenger living or sleeping quarters or areas with electric conduits or wires have to be insulated to a considerable degree. This was usually done with asbestos panels. Gaskets were used around doors, and for this a "Spread Limpitus" installation containing asbestos was employed. On this basis, it was suggested it is quite possible that a number of people, other than installers and insulators, who had been working in the areas where the panels and insulation were installed and who, therefore, could have come into contact with asbestos fibres, particularly in the areas where panels with a high asbestos content were cut and sanded.



A. ASSISTANCE PROGRAMME FOR ELLIOT LAKE URANIUM MINERS - JULY, 1975

On May 6, 1975, the Honourable John MacBeth, Q.C., Minister of Labour (then), issued a statement to the effect that Government of Ontario had adopted a number of measures to assist those workers who have been adversely affected by exposure employment to silica dust and radiation in the Elliot Lake uranium mines, and that these measures will also have impact on an industry-wide basis.

In essence, the statement covered the following points:

- Workers with silicosis will be encouraged to leave exposure employment and will be assisted with compensation based on difference in earnings and through rehabilitation retraining and resettlement.
- Workers with significant dust effects but who are not disabled by silicosis will be given rehabilitation assistance including retraining and resettlement grants.
- Re-employment in other low risk mines will be encouraged by guaranteeing that such employers will not be charged with the cost of future chest disease claims for these rehabilitated workers.
- Workers who experience a total of four working level months of radiation exposure in one year and leave exposure employment for the balance of the year will be eligible for compensation for temporary partial disability until they return to exposure employment.
- Workers with lifetime radiation exposure of over one hundred and twenty working level months will be eligible for rehabilitation assistance on the same basis as workers with significant dust effects.

On July 22, 1975, the Board resolved that it would consider claims on the basis outlined in the Minister's announcement in the Legislature, but that it would not be responsible for the establishment of exposure standards or for monitoring of the workers.

The rationale for the Special Programme for workers in mineral dust was based on the premise that for those workers showing "incipient" signs of pneumoconiosis (dust effects), removal from further exposure would possibly prevent progression of the condition to manifest disease and possible impairment. For those with manifest disease, but without impairment (being therefore ineligible for compensation), removal from further exposure might be expected to retard progression and development of subsequent impairment.

The definition of "incipient" mineral dust disease will relate to radiological change and/or pulmonary function impairment not of a degree to warrant a diagnosis of pneumoconiosis, although of such a nature, that in others have been known to precede the development of manifest disease.





The validity and the usefulness of this approach depends on several factors.

- The intensity and duration of the prior exposure.
- The nature and intensity of the present exposure.
- The characteristics of the pneumoconiosis and the associate dose response relationships found in the work place.
- The working population.

#### B. EXTENSION OF THE INITIAL ASSISTANCE PROGRAMME - OCTOBER 1975

On October 8, 1975, the Board reviewed the results of the assistance programme for the Elliot Lake miners, approved certain modifications in it and resolved that the principles of this programme would also apply:

- in other parts of the province
- to workers in mining, glass, steel and other industries showing silica dust effects diagnosed as a "4" rating, or silicosis diagnosed as a "5" rating, and
- for other lung disease entities such as disease caused by employment exposure to asbestos.

The implementation of the Special Programme was never conceived to be completely dependent upon dust free environments. The Ministry of Labour Directive of May 6, 1975 specifically provided for low risk mines as alternative work locations for miners with incipient silicosis or for those with silicosis (without significant impairment). The free silica levels in the respirable dust in these mines were not expected to add significantly to the ongoing risk.

In the case of asbestos, it is by no means agreed that withdrawal from exposure will stop or slow progression of asbestosis.<sup>20</sup>

Parkes states that mild to moderate fibrosis in asbestosis is related to the dose retained and an increase in dose does not seem to cause further progression - it seems to be due to other factors.<sup>21</sup>

In incipient asbestosis, the problems of early detection are complex.

<sup>20</sup> Task Force on Occupational Respiratory Disease (Pneumoconiosis), February, 1979.

<sup>21</sup> W.R. Parkes, "Asbestos Related Disorders", British Journal Disease of Chest, Volume 67, Page 261, 1973.





In the opinion of Sluis-Cremer<sup>22</sup>, "the study indicates there is no way to pinpoint those with minimal asbestosis in life in order to remove them from further exposures early."

The same report suggested "...physiological test in this series do not indicate persons who might have early asbestosis with normal chest x-rays."

The identification of incipient asbestosis depends on early detection of dust effects (without symptomatology). While periodic comparative chest radiographs or pulmonary function tests are likely to improve chances of detection, Becklake states that

"the radiologic changes are invariably of a non-specific nature similar to those occurring with aging or the cigarette habit, hence the opinion that in an individual case they reflect fibrosis, should be guarded."<sup>23</sup>

Even in those workers with long term dust exposure (with no evidence of x-ray or pulmonary function change and no clinical disease), showing only decreased pulmonary compliance and increased upstream resistance, - Becklake observed,

"it remains to be shown that these will progress to asbestosis or that any intervention such as removal from exposure would prevent the ultimate development of disease."<sup>23</sup>

<sup>22</sup> G. K. Sluis-Cremer, "Asbestosis in South African Asbestos Miners," Environmental Research, Volume 3, Pages 310-319, 1970.

<sup>23</sup> Margaret Becklake, "State of the Art - Asbestos Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," American Review of Respiratory Disease, Volume 114, 1976.



C. ASSISTANCE PROGRAMME FOR ASBESTOS WORKERS - MAY 1976

a) Introduction

After the assistance programme for the Elliot Lake miners had become operative, the Asbestos Workers' Union requested the Board to establish a similar programme for asbestos workers. This task was undertaken by the Rehabilitation Services Division (then) and the Claims Services Division in October 1975, and a programme, containing the same benefits as the Elliot Lake miners' programme, for workers suffering from asbestosis and asbestos fibre dust effects, was approved by the Board on May 11, 1976.

b) Assistance Programme for Asbestos Workers - Details

1. Eligibility - Under this programme, asbestos workers eligible for assistance will be those who suffer from:

(a) Asbestosis

(b) Asbestos Fibre Dust Effect (AFDE).

(See medical guidelines, pages 1-4)

Before contact is established with the employee, the Chest Disease Consultant, Medical Branch, will confirm the diagnosis according to the approved medical criteria.

2. Initial Approach (Interviews and Documentation) - A team consisting of Medical Branch, Vocational Rehabilitation Branch and Claims Adjudication Branch representatives will initially interview asbestos workers eligible for the programme and explain the benefits to them.

3. Employee/Employer Confidentiality - The employer will not be notified of the names of the AFDE cases who request an interview. However, the employer will be aware of any asbestosis and AFDE employees who have already filed a claim or are in receipt of a pension.

If a Vocational Rehabilitation retraining/placement programme is started for an asbestos worker, his employer will have to be notified as a claim file must be set up at that time.

4. Representation - An asbestos worker may have a representative present during the team interview.

5. Vocational Rehabilitation Assistance -

(a) Work Adjustment Rehabilitation Allowance up to the equivalent of full compensation, while the worker is seeking work in a new location, may be paid up to a twelve month period with review at the end of that time.



- (b) Travel expenses will be paid for the worker and his spouse in order to take a preliminary trip to investigate the area in which they wish to relocate, if required.
- (c) The assistance will also include moving expenses, if necessary, for successful rehabilitation (see page 61 re Relocation Expenses).
- (d) Should the worker decide to relocate outside the Province of Ontario, he will be advised that the Vocational Rehabilitation Branch will be limited in the assistance which it can furnish to him. (Contact would be made with the appropriate Rehabilitation Branch of other Boards in such cases, and a request would be made for assistance as required).
- (e) Where a worker transfers from a dust exposure area to non-exposure employment with the same employer or new employer, wage difference may be paid under Section 53 for a period up to twelve months with review at the end of that time.
- (f) Retraining of worker for suitable non-exposure employment.
- (g) Payment of wage difference for a period up to twelve months after completion of retraining with review at the end of that time.

6. Identification of Future AFDE Cases - Selected chest films taken of asbestos workers by the Ministry of Health will be reviewed by the Chest Disease Consultant, Medical Branch, to identify cases of Asbestos Fibre Dust Effect.

7. Payment of Work Adjustment Rehabilitation Allowance - This is payable under the supervision of the Board's Vocational Rehabilitation Branch and benefits are conditional on the asbestos worker being available for and cooperating in seeking alternate employment and/or retraining. Relocation allowances are also payable if the worker moves to another community to accept employment or retraining.

8. Asbestos Fibre Dust Effect Cases (AFDE) -

- (a) Working With Wage Loss - Benefits paid will be based on earnings loss on new job when compared with earnings in exposure employment prior to job change. These benefits are payable for up to twelve months subject to review at the end of that period and will cease if the employee's non-exposure work wage is no longer less than his exposure earnings.
- (b) No Job and Seeking Employment - The equivalent of up to full compensation benefits up to the statutory maximum will be paid for one year with review at the end of that period. (Compensation rate calculated on last year of exposure earnings).

9. Benefits in Asbestosis Cases

The same as provided under point 8 (a or b) above. An asbestos worker in receipt of a pension for impairment is entitled to receive the pension in addition to the wage loss benefits payable under this programme.





10. Retraining Programme - Where the asbestos worker is placed on a retraining programme by the Board's Vocational Rehabilitation Branch he shall receive full Rehabilitation benefits for the duration of the retraining programme. Payment of wage difference for a period up to twelve months after completion of retraining will be considered. All Work Adjustment Rehabilitation Allowances are subject to review on expiry.

D. INSTRUCTIONS (APPROVED BY THE BOARD MAY 11, 1976) FOR CLAIMS  
ADJUDICATION BRANCH FOR THE PROCESSING OF CLAIMS UNDER  
THE ASSISTANCE PROGRAMME FOR ASBESTOS WORKERS

1. Work Adjustment Rehabilitation Allowance

This type of payment is paid in the following cases:

- a) Asbestosis
- b) Asbestos Fibre Dust Effect (AFDE).

When considering payment of benefits to make up for wage loss where the employee has moved out of exposure to another type of work, we should obtain the following information:

- The Worker's Gross Earnings in Exposure Employment for One Year Prior to the Job Change. When considering payment of benefits, a Form 9E or EA2 should be sent out every four weeks in order to obtain the worker's current earnings for the four week period. The Work Adjustment Rehabilitation Allowance will then be paid, calculated in the same manner as temporary partial difference.
- This Payment Can Be Continued For One Year And At The End Of The Time Should Be Reviewed. Payment mark-up should be made on the Form 712 indicating "pay out of Rehabilitation Section 53 funds."

The file jacket at the top should be stamped "AFDE Programme" and the summary sheet stamped at the bottom with the same notation.

2. Training Programme

Workers retraining under this programme will be paid exclusively by the Rehabilitation Branch under Section 53. However, the Claims Adjudicator should obtain the gross earnings for one year prior to the start of the training program (last year of exposure), on which the Rehabilitation Branch will base its payments.

After the completion of retraining, if the worker is suffering a wage loss, consideration can be given to paying the Work Adjustment Rehabilitation Allowance up to a twelve month period.



3. Left Exposure and Looking for Employment

This type of case will be serviced by the Vocational Rehabilitation Branch and the worker will receive the equivalent of temporary total disability.

Earnings for one year prior to leaving exposure will also be required in this case.

4. Old Cases Where Pensions Are Being Paid

Any workers coming under this programme who are currently in receipt of a pension for asbestosis are entitled to the pension in addition to the wage loss benefits payable under this programme.

5. Permanent Disability Awards

Pensions for asbestosis are not deducted from the benefits payable under this programme.

E. SPECIAL REHABILITATION ASSISTANCE PROGRAMMES TO REMOVE WORKERS FROM HAZARDS OF INDUSTRIALLY GENERATED DISEASES - JUNE 15, 1977

a) Background

Following approval of the Special Rehabilitation Assistance Programmes for Elliot Lake Uranium Miners (in 1975) and an identical programme for asbestos workers (in 1976), the Board directed that one policy document be developed for:

- miners with silica particle dust effects with "4" or "5" (x-ray) ratings
- uranium miners with radiation exposure in excess of 120 working level months
- workers with asbestosis or asbestos dust effects, and
- for other industrial hazards that may be recognized in the future.

On this basis, the information in the previously approved policy documents was amalgamated into one policy document devoid of any procedural items, but without any other changes in the provisions of the programmes. The resulting policy document "Special Rehabilitation Assistance Programmes To Remove Workers from the Hazards of Industrially Generated Diseases", which was approved by the Board on June 15, 1977, now applied not just to the uranium miners in Elliot Lake and workers exposed to asbestos dust but also to other segments of the workforce and other industrial hazards, as they become identified.



b) Details of the Programme

1. Eligibility - The following classifications of workers are eligible for assistance:

- 1.1 Miners with "4" ratings (silica particle dust effect in x-ray) and with "5" ratings (silicotics with or without a rated disability).
- 1.2 Uranium miners with radiation exposure in excess of 120 working level months.
- 1.3 Employees with asbestosis or asbestos fibre dust effects (AFDE).
- 1.4 Employees affected by exposure in employment, including:
  - 1.4.1 Employees with "4" ratings and "5" ratings in other industries.
  - 1.4.2 Employees with mineral dust effects (MDE) as identified.
  - 1.4.3 Other employees as identified and approved for the programme.

2. Employee/Employer Confidentiality -

- 2.1 The employer will not be notified of the names of employees who qualify for the programmes and who request interview.
- 2.2 The employer will already be aware of any employees who have filed a claim or are in receipt of a pension.
- 2.3 If a Vocational Rehabilitation Programme is instituted for an affected employee, the employer shall be notified since a claim file must be set up at that time.

3. Representation -

- 3.1 The employee may have a representative of his choice present during the team interview.

4. Extended Benefits Assistance Programme -

- 4.1 Rehabilitation assistance including training, payment of full compensation or partial compensation on a wage loss basis, paid from Section 53 shall be provided. The assistance shall also include moving expenses to anywhere in Canada if, in the opinion of the Vocational Rehabilitation Branch, it is reasonable to expect that this will result in suitable employment.





- 4.2 Approved travelling expenses shall be paid for the employee and his spouse in order to take a preliminary trip to investigate the area in which they wish to relocate.
  - 4.3 Rehabilitation Assistance Allowance equivalent to full compensation and in accordance with current Vocational Rehabilitation Policy shall be paid. This shall apply so long as the employee is cooperating with the Vocational Rehabilitation Branch and actively seeking work in the new location.
  - 4.4 Should relocation outside the Province of Ontario be approved by the Vocational Rehabilitation Branch, the employee shall be advised that the assistance which can be furnished will be limited. The Board's Vocational Rehabilitation services apply only in Ontario. Contact would be made with the appropriate Rehabilitation Branch of other Boards in such cases and a request would be made for whatever assistance they are able to provide.
  - 4.5 Relocation expenses shall be considered to include normal moving expenses, transportation costs for the employee and family, cost of pre-inspection trip, lease assistance and/or sale of property assistance, a portion of legal fees, and a \$500.00 grant for incidental costs.
  - 4.6 Before compensation on a wage loss basis shall be paid under Section 53 where an employee transfers from exposure employment to non-exposure employment such transfer must be approved by the Vocational Rehabilitation Branch. This transfer of employment must be considered by the Rehabilitation Services Division to be physically suitable, vocationally appropriate and economically sound. Payments will be made as long as the employee continues in such employment based upon the difference between his current earnings and his earnings at the time of leaving exposure employment.
  - 4.7 All decisions relating to this programme are subject to the basic right of appeal.
5. Identification of Employees -
- 5.1 The Board is made aware of current status of miners by submission of Form 86 (this includes the x-ray rating) to the Board's Programme Planning and Statistical Branch. Form 86 originates from the Ministry of Health Chest Examining Stations.
  - 5.2 Uranium miners are specifically identified and copies of Form 86's are directed to the attention of the Board's Chest Disease Consultant. This allows interception of all "4" and "5" ratings from uranium mining operations (Form 86 originates from the Ministry of Health Chest Examining Stations).



- 5.3 To further enhance identification of cases, all x-ray films on "4" ratings shall be submitted to the Board's Chest Disease Consultant when such a rating is initially proposed. A current list of all such ratings shall be maintained by the Chest Disease Consultant, Medical Branch and the Programme Planning and Statistical Branch. (X-ray films originate from the Ministry of Health Chest Examining Stations.)
- 5.4 Employees who have accumulated 120 working level months (WLM) of radiation in employment shall be identified by the designated Ministerial Authority and such identification shall be directed to the attention of the Board's Chest Disease Consultant.
- 5.5 When the appropriate Ministry responsible for the operation of the Industrial Chest Disease Service identifies an employee with asbestos fibre dust effect (AFDE) the Board's Chest Disease Consultant shall be notified and x-ray films shall be submitted to the Board's Chest Disease Consultant on request.
- 5.6 As new groups at risk are identified, suitable guidelines shall be developed and incorporated into this policy document.

c) Special Rehabilitation Assistance Programme  
For Johns-Manville Canada Inc. Workers

The usefulness of and the difficulties encountered in applying the Special Programme to asbestos workers in general and to Johns-Manville in particular must be evaluated in the light of these relationships and observations. The average duration of exposure in those selected for the Programme approached 25 years, and the mean index of exposure (using the British Occupational Hygiene Society's formula - fibre/c.c./years) would likely lie between 150 and 300 fibres/c.c./years.

0.5 fibres/c.c. was chosen as the threshold limit for the Programme. In those working areas with the majority of counts above 0.5, workers with asbestos fibre dust effects (AFDE) or asbestosis would qualify. Only the transite pipe production department fell into this category. In all other areas, the counts were below 0.5 fibre/c.c. and most between 0.1 and 0.3 fibres/c.c.

Using 200 fibres/c.c./years as an average for those interviewed, ongoing exposure (additional three to eight years) might mean an added load of one to three fibres/c.c./years.

The significance of this additional risk must be set against the difficulties and limitations in the successful retraining, relocating, or upgrading of men reaching the end of their working careers, most with no trade or experience beyond that acquired with their employer.



Special Rehabilitation Assistance Programme

For

Johns-Manville Canada, Inc.

Statistics As At December 31, 1980

	<u>Total</u>	<u>Percentage</u>
A. <u>Total Known Asbestosis &amp; AFDE Cases</u>	<u>80</u>	<u>100.00%</u>
Breakdown:		
1. Asbestosis	31	38.75%
2. AFDE	48	60.00%
3. Unclassified	<u>1</u>	<u>1.25%</u>
TOTAL	<u>80</u>	<u>100.00%</u>
B. <u>Total Interviewed by The Team</u>	67	83/75%
Total not interviewed by Team (includes cases previously active with V.R.D., also no shows and four (4) since deceased).	<u>13</u>	<u>16.25%</u>
TOTAL	<u>80</u>	<u>100.00%</u>
C. <u>Exposure vs. Non-Exposure</u>		
1. Total out of exposure at the team interviews	37	46.25%
2. Total in exposure at time of team interviews	38	47.50%
3. Unclassified at time of team interviews	1	1.25%
4. Deceased at time of team interviews	<u>4</u>	<u>5.00%</u>
TOTAL	<u>80</u>	<u>100.00%</u>
D. <u>Participation vs. Non-Participation</u>		
1. Total electing to participate	18	22.50%
2. Total electing not to participate	36	45.00%
3. Total undecided	16	20.00%
4. Total subsequently deciding to withdraw (change in circumstances, reversal of former decision, etc.)	--	---
5. Unclassified	--	---
6. Deceased	<u>10</u>	<u>12.50%</u>
TOTAL	<u>80</u>	<u>100.00%</u>





	<u>Total</u>	<u>Percentage</u>
<u>E. Qualified for Entry vs. Non-Qualification in Terms of Hazardous vs. Non-Hazardous Employment at Time of Team Interviews</u>		
Total number above who did not qualify as not in hazardous employment at time of team interviews	48	60.00%
Total number above who did qualify as in hazardous employment at time of team interviews	<u>32</u>	<u>40.00%</u>
TOTAL	<u>80</u>	<u>100.00%</u>
<u>F. Acceptance vs. Refusals</u>		
Total number who did not qualify	48	60.00%
Total number (above) who refused programme outright	22	
Total number who qualified	32	40.00%
Total number (above) who refused programme outright	14	
Total number who did participate in programme	<u>18</u>	
TOTAL	<u>80</u>	<u>100.00%</u>
<u>TOTAL ACTIVE CASELOAD</u>		
Breakdown:		
1. (a) In exposure	Nil	
(b) Out of exposure	33	
(c) Unclassified	<u>--</u>	
TOTAL	<u>33</u>	
2. (a) Out of exposure participating and receiving T.P.D. or pension or combination	18	
(b) Off due to compensable reasons	Nil	
(c) Off due to non-compensable reasons	8	
3. On Training		
(a) Formal	3	
(b) Training-on-the-job	7	
4. Relocating	3	



d) Relocation Expenses to Workers Under the Special  
Rehabilitation Assistance Programmes to Remove  
Workers from the Hazards of Industrially Generated  
Disease - May 1976

---

The following provisions apply to workers under the above programmes who are eligible to relocation and are required to move. (These criteria were originally approved by the Board in May 1975 for eligible Elliot Lake miners and subsequently extended to eligible asbestos workers in 1976.)

1. The normal expenses for the movement of household effects, subject to a tender from two moving firms.
2. The normal cost of transporting the employee and his family.
3. (a) Cost of one pre-inspection trip to home purchase area for wife and husband.  
  
(b) Cost of return travel within Ontario once per week up to six weeks when there is an unavoidable delay in moving family.
4. Residential property:
  - (a) One of the following items may be allowed:
    - i) Discharge of lease (overlapping rent)
    - ii) Sale of residence:
      - legal fees up to 1% of sale price
      - realty fee, local tariff or M.L.S. cost
    - iii) Moving a mobile home:
      - cost of moving
      - discharge of lease.
  - (b) Legal fees on purchase, not exceeding 1½% of purchase price. This is payable only if the employee was entitled to claim under (ii) above (sale of residence).
5. Grant of \$500.00 to cover incidental costs of moving.



C O P Y

DEPARTMENT OF EPIDEMIOLOGY AND HEALTH  
3773 University Street, Montreal, Que., Canada, H3A 2B4

McGILL UNIVERSITY

Dr. Cameron C. Gray  
Executive Medical Director  
Ontario Thoracic Society  
157 Willowdale Ave.  
Willowdale, Ont. M2N 4Y7

December 19th, 1974.

Dear Cam,

Enclosed you will find the reprints you requested: I hope they will be helpful.

I shall try to answer your other questions which are, of course, all difficult ones though I have fairly firm views on them.

- 1) I think there is adequate evidence that in smokers asbestos related bronchial cancer can develop in the absence of much in the way of asbestosis. Thus the Quebec Compensation Commission has adopted what seems to me a fair benefit-of-the-doubt position in compensating Bronchial Ca requiring only that the subject has had heavy exposure (about 400 mppef-yrs.); evidence of lung fibrosis e.g. a few pleural plaques is also helpful though not mandatory. Obviously some cigarette lung cancers will be included but there is no way to reasonably exclude these, hence the benefit-of-the-doubt decision.
- 2) In compensation terms, disability is what prevents gainful employment, hence exercise testing is essential. Impairment for compensation purposes should in my view consist of complete resting lung function tests and a Stage I and Stage II exercise test of the Norman Jones variety. Resting lung functions (volumes, flows,  $D_{CO}$  and mechanics, the latter being occasionally indicated) should be regarded as descriptive measurements which do not give direct information on lung gas exchange on exercise from mild to maximal; this must be known in order to evaluate disability. Indeed, as you and I know from experience, with the lung's large reserve of function, exercise capacity can be considerably reduced in the absence of anything definite in static lung function measurements, normal values for which are only accurate 10 to about 20%. Hence complete rest and graded exercise tests seem to me obligatory for a disability evaluation. This is done by the Quebec Workmen's Compensation Commission.





Dr. Cameron C. Gray  
Willowdale, Ont.

December 19th, 1974.

3) The study on lung function pattern in asbestos workers to which you referred is the subject of Dr. Fournier's thesis and will, we hope, be published next year. We found that obstructive function profiles occur with somewhat greater frequency than restrictive profiles in asbestos workers who smoke, and that an obstructive profile is at least as common as a restrictive profile in the presence of radiological asbestosis. This means, I believe, that both profiles should be accepted as associated with asbestos exposure - in other words, I do not support the view that obstruction when present is attributable to other factors, e.g. cigarettes. I will certainly mail you a copy of this paper when ready.

I hope this information may be of some help to you. Please write again if I have not answered some of your questions satisfactorily.

Yours sincerely,

Margaret R. Becklake, M.D.

Enclosures

mr/b/cm



ASBESTOS DISEASE - INTERIM REPORT, 1975

DR. A. C. RITCHIE, PROFESSOR OF PATHOLOGY, UNIVERSITY OF TORONTO

The time seems to have come to reconsider the relationship between asbestos and disease. New information is appearing which makes it desirable to reconsider some of the positions we have held until now.

1. Four kinds of asbestos are important in causing disease; Chrysotile, Crocidolite, Amosite and Anthophyllite.
2. Chrysotile is the form mined in Canada and the principle form used here, though some Crocidolite is imported, and other fibres are occasionally used.
3. The pathogenicity of the various types of fibre probably varies. Crocidolite is probably the most dangerous, and Chrysotile the least. Data about Amosite suggest it is intermediate, but little is known about Anthophyllite.

Cooper, 1967  
Editorial, Lancet, 1973  
Enterline et al, 1972  
Enterline & Henderson, 1973  
Gross & Harley, 1973  
Harrington et al, 1971  
Selikoff, Hammond & Churg, 1972  
Sleggs et al, 1961  
Smith, Miller & Churg et al,  
1965

Smith, Miller, Elasser et al, 1965  
Stanton & Wrench, 1972  
Wagner, 1962  
Wagner, 1970  
Wagner & Berry, 1969  
Wagner et al, 1971  
Wagner & Skidmore, 1965  
Wagner et al, 1960  
Wright, 1969

4. The pathogenicity of asbestos depends on several factors in addition to its type. The physical form of the fibre, in particular its diameter and its length, seem of major note, though information is incomplete.

McCullagh, 1974  
Stanton & Wrench, 1972  
Stanton, 1974

Timbrell et al, 1971  
Wagner et al, 1971

5. Some evidence suggests that other fibres, such as glass, may be similarly pathogenic if of similar size, though again the evidence is incomplete.

Stanton & Wrench, 1972  
Stanton, 1974



6. The proteinaceous covering of asbestos in asbestos bodies may reduce or prevent their pathogenicity. Chrysotile may be more easily coated than other fibres.

Botham & Holt, 1971  
Thomson, 1966

7. Oils, trace metals and hydrocarbons such as Benzpyrene adsorbed on the asbestos fibres do not seem important in causing disease.

Cooper, 1967	Langer et al, 1972
Dixon et al, 1970	Stanton & Wrench, 1972
Gross et al, 1967	Wagner, 1962
Gross & Harley, 1973	Wagner, 1970
Harington, 1962	Webster, 1973
Harington, 1965	World Health Organization, 1973
Harington & Roe, 1965	Wright, 1969

8. A number of methods of isolating and enumerating asbestos fibres in asbestos bodies in the lung and other tissues have been described.

Anjilvel et al, 1966	Gross, deTreville et al, 1968
Ashcroft, 1968a	Langer et al, 1973
Ashcroft and Heppleston, 1973	Langer et al, 1971
Badollet & Ganttt, 1965	Naylor, 1974
Berkley et al, 1965	Plowman, 1973
Bignon et al, 1970	Rosen et al, 1972
Cauna et al, 1965	Smith & Naylor, 1972
Gold, 1967	

9. Techniques for the isolation of asbestos fibres from air or fluids have been detailed.

Bartosiewicz, 1973	Mitchell, 1961
Biles, 1968	Rickards & Badami, 1971
Cunningham & Pontefract, 1973	Selikoff, Nicholson & Langer, 1972
Holt & Young, 1973	

10. Asbestos is ubiquitous in our environment. Several studies have shown it present in nearly 100% of lungs from patients coming to autopsy. Males show more fibres per gram of lung than do females (Table A, page 16).

Angilvel et al, 1966	Roberts, 1967
Ashcroft, 1968a	Rosen et al, 1972
Bignon et al, 1970	Selikoff & Hammond, 1968
Cauna et al, 1965	Selikoff, Nicholson & Langer, 1972
Editorial, JAMA, 1966	Smith & Naylor, 1972
Elmes et al, 1965a	Thomson, 1965
Hagerstrand et al, 1968	Thomson & Graves, 1966
Hagerstrand & Seifert, 1973	Thomson et al, 1963
Langer et al, 1971	Utidge et al, 1968
Meurman, 1966	Wagner et al, 1968
McPherson & Davidson, 1969	Warnock & Churg, 1975
Penman & Thomson, 1970	Wright, 1969
Polliack & Sacks, 1968	Xipell & Bhathal, 1969





11. Different kinds of asbestos are present in the lung, with Chrysotile the most common. Degradation of the fibres is evident.  

Langer et al, 1971	Selikoff, Nicholson & Langer, 1972
Langer et al, 1972	Warnock et al, 1975
12. Those with occupational exposure to asbestos have much larger quantities of asbestos in their lungs than has the general population, though quantitation is inexact.  

Ashcroft & Heppleston, 1973	Thomson, 1965
Beattie & Knox, 1961	
13. There is a suggestion not yet confirmed that asbestos fibres may be more numerous in patients with cancer than in others.  

Utidjian et al, 1968  
Warnock & Churg, 1975
14. Asbestos may contaminate the environment around an asbestos plant, or a worker may carry home asbestos to contaminate his house. Animals may be carriers.  

Ashcroft, 1968	Navratil, 1971
Bohlig et al, 1970	Navratil & Trippé, 1972
Borow et al, 1967	Newhouse & Thompson, 1965a
Dalquen et al, 1969	Newhouse & Thompson, 1965b
Laamanen et al, 1965	Selikoff et al, 1967
Lieben & Pistawka, 1967	Webster, 1963
McEwen et al, 1972	World Health Organization, 1973
15. Asbestos in the lung tends to migrate to or collect at the base of the lower lobes, particularly within a centimeter or so of the diaphragmatic pleura, or in relation to respiratory bronchioles.  

Gross, 1966	Thomson, 1966
Thomson, 1962	Timbrell, 1965
Thomson, 1965	Wagner et al, 1965
16. In the lung, asbestos remains for long periods, much more than 20 years, though there is evidence that some of it may be fragmented or perhaps lost.  

Beattie & Knox, 1961	Nagelschmidt, 1965
Lynch & Cannon, 1948	
17. A minority of the asbestos fibres in the lung form asbestos bodies. Most remain uncoated, and much as small, short fragments.  

Ashcroft & Heppleston, 1973	Davis, 1965
Bignon et al, 1970	



18. Asbestos bodies are formed as asbestos fibres, are enclosed in whole or part in macrophages, which invest them with a proteinaceous coat, which becomes impregnated with iron and other materials.

Beattie, 1961	Gloyne, 1930
Botham & Holt, 1968	Lynch, 1937
Botham & Holt, 1971	Stewart, 1928
Botham & Holt, 1972	Stewart & Haddow, 1929
Cooke, 1929	Suzuki & Churg, 1969
Davis, 1965	Suzuki & Churg, 1970
Gaensler & Addington, 1969	Williams, 1934
Gloyne, 1929	

19. Asbestos bodies become segmented as they age, and break at the narrowed places, exposing again the asbestos fibre.

Beattie, 1961	Langer et al, 1972
Botham & Holt, 1968	Suzuki & Churg, 1970

20. Asbestos bodies may be becoming more numerous in the lung.

Um, 1971

21. Asbestos bodies are highly characteristic of inhaled asbestos, but similar bodies are occasionally formed around other fibres, and many asbestos fibres in the tissues remain uncoated.

Ashcroft, 1968b	Gross, 1968
Botham & Holt, 1971	Gross, deTreville et al, 1968
Cooper, 1967	Suzuki & Churg, 1970
Gaensler & Addington, 1969	Wright, 1969

22. Exposure to asbestos has been associated with rheumatoid disease in the lung, or an increase in rheumatoid factor in the blood.

Pernis et al, 1965	White et al, 1974
Rickards & Barret, 1958	

23. Fibrosis of the lung was the first lesion to be associated with asbestos, and fibrosis so caused is called Asbestosis.

Ashcroft, 1968 (Tyneside)	Moigneteau et al, 1974
Bonser et al, 1955 (Carlisle)	McPheeters, 1936
Cartier, 1955 (Quebec)	McPherson & Davidson, 1969
Cooke, 1927 (Wigan)	McVittie, 1965
Donnelly, 1933 (Carolina)	Nagelschmidt, 1965
Dreesen et al, 1938 (Washington)	Newhouse et al, 1972
Egbert & Geiger, 1936 (Yale)	Roberts, 1967
Elmes, 1966 (Belfast)	Selikoff et al, 1967
Enterline, 1965 (Washington)	Selikoff et al, 1964
Enterline et al, 1972 (Washington)	Selikoff et al, 1965a
Enterline & Kendrick, 1967 (Quebec)	Selikoff, Hammond & Churg, 1972
	Sluis-Cremer, 1965
	Sluis-Cremer & Theron, 1965



Feil, 1931 (France)	Smith, 1955a
Gardner, 1938 (Saranac Lake)	Smith, 1955b
Hammond et al, 1965 (New York)	Smith, 1952
Harries et al, 1973 (review)	Stewart et al, 1931
Hinson, 1965 (review)	Stewart, 1928
Knox et al, 1965 (Rochdale)	Wegelius, 1947
Knox et al, 1968 (Rochdale)	Wood & Gloyne, 1930
Lanza et al, 1935	Wood & Gloyne, 1931
Merewether, 1930 (U.K.)	Wood & Gloyne, 1934
Merewether, 1933 (review)	

24. The fibrosis in Asbestosis involves primarily the lower lobes, and particularly their bases. It is at first patchy and interstitial, but with accentuation around bronchioles. Later it may become extensive and obliterative. Honeycombing may follow.

Caplan et al, 1965	Heard & Williams, 1961
Ellman, 1933	Kuhn & Kuo, 1973
Ellman, 1940-41	McDonald, 1927
Gough, 1965	

25. Asbestosis as revealed by x-ray occurs only after a latent period rarely less than 5 years, usually in excess of 10 years and often longer.

Ellman, 1933	McPheeters, 1936
Ellman, 1940-41	Selikoff et al, 1967
Isselbacher et al, 1953	Selikoff et al, 1965a
Merewether, 1930	Smith, 1955a
Murphy et al, 1971	Wright, 1969

26. Asbestosis may develop long after exposure has been terminated. Asbestos remains at least in large part once it is in the lung.

Selikoff et al, 1967

27. Asbestosis develops only in those exposed to relatively high concentrations of dust. Minor occupational exposure brings little or no increase in the incidence of pulmonary fibrosis. Duration of exposure is also important.

Beattie & Knox, 1961	Merewether, 1930
Cooper, 1967	Murphy et al, 1971
Fleischer, 1946	Selikoff et al, 1967
Glover, 1973	Wright, 1969
Knox et al, 1965	

28. Reducing the dust exposure has prevented or almost prevented Asbestosis.

Knox et al, 1965	Wright, 1969
Knox et al, 1968	





29. A relationship between the uptake of asbestos by macrophages and the production of fibrosis has been suggested but not proved. Recently it has been proposed that very small fibres may damage the macrophages as silica may do, causing them to release a factor which causes fibrosis.  
  
Beattie, 1961  
Beattie & Knox, 1961  
Davis, 1963  
Davis, 1965  
Davis, 1972 a & b  
Holt et al, 1964  
  
Holt et al, 1965  
Miller et al, 1975  
Nagelschmidt, 1965  
Rajan et al, 1972  
Richards & Morris, 1973  
Wagner, 1965
30. All types of asbestos fibre may cause Asbestosis.  
  
Selikoff, Hammond & Churg, 1972  
World Health Organization, 1973
31. No correlation between the physical character of the Asbestosis and the risk of Asbestosis has been established.
32. Pulmonary function studies may be abnormal before there is radiological evidence of Asbestosis. Reduced vital capacity and increased resistance in small airways are early signs. Diffusing capacity is often reduced.  
  
Bader, Bader & Selikoff, 1961  
Bader et al, 1970  
Bader et al, 1965  
Becklake et al, 1972  
Gaensler & Kaplan, 1971  
Gernex-Rieux et al, 1954  
  
Heard & Williams, 1961  
Hunt, 1965  
Jodoin et al, 1971  
McCullagh, 1974  
Selikoff et al, 1967  
Thomson et al, 1965
33. At least in some parts of the world, exposure to asbestos is associated with an increased incidence of pleural plaques, which are often calcified.  
  
Bohlig, 1965 (Hamburg)  
Eisenstadt, 1962 (Texas)  
Hagerstrand & Seifert, 1973 (Malmö)  
Hourihane et al, 1966 (London)  
Kiviluoto, 1960 (Finland)  
Kiviluoto, 1965 (Finland)  
Lynch & Cannon, 1948 (Carolina)  
Neurman, 1966 (Finland)  
Meurman, 1968 (Finland)  
  
Navratil, 1971 (Prague)  
Navratil & Trippé (Prague)  
Raunio, 1966 (Finland)  
Roberts, 1967 (Glasgow)  
Roberts, 1971 (Glasgow)  
Rosen et al, 1973 (New York)  
Selikoff, 1965 (New York)  
Selikoff et al, 1967 (New York)  
World Health Organization, 1973  
Wright, 1969 (review)
34. The pleural plaques alone do not cause disability.  
  
Burlikov & Michailova, 1970  
Kiviluoto, 1960



35. They may be found in increased incidence in populations living around asbestos plants, or in parts of the world in which natural deposits of asbestos are abundant.
- |                             |                   |
|-----------------------------|-------------------|
| Burilkov & Michailova, 1970 | Kiviluoto, 1965   |
| Hourihane et al, 1966       | Raunio, 1966      |
| Kiviluoto, 1960             | Zolov et al, 1967 |
36. Asbestos has rarely been demonstrated in pleural plaques, but may be found in the lung of these patients.
- |                             |                   |
|-----------------------------|-------------------|
| Gaensler & Kaplan, 1971     | Roberts, 1971     |
| Hagerstrand & Seifert, 1973 | Rosen, 1974       |
| Meurman, 1966               | Rosen et al, 1973 |
37. Pleural plaques believed due to asbestos may occur without fibrosis in the underlying lung.
- |                  |                       |
|------------------|-----------------------|
| Eisenstadt, 1962 | Hourihane et al, 1966 |
| Eisenstadt, 1965 | Meurman, 1966         |
38. Similar plaques may be caused by other means.
- |                       |                                 |
|-----------------------|---------------------------------|
| Hourihane et al, 1966 | Rous & Studeny, 1970            |
| Meurman, 1968         | Smith, 1952                     |
| Navratil, 1971        | World Health Organization, 1973 |
| Robinson, 1972        | Wright, 1969                    |
39. Pleural effusion may be due to exposure to asbestos, though this is a rare cause of effusion.
- |                         |                       |
|-------------------------|-----------------------|
| Berger & Mejia, 1973    | Leuallen & Carr, 1955 |
| Eisenstadt, 1964        | Roper & Waring, 1955  |
| Eisenstadt, 1965        | Sochocky, 1966        |
| Gaensler & Kaplan, 1971 |                       |
40. Mesothelioma of the pleura or peritoneum was once considered rare, but is now recognized as a well defined neoplasm.
- |                          |                              |
|--------------------------|------------------------------|
| Bolio-Cicero et al, 1961 | McDonald, Magner et al, 1973 |
| Campbell, 1950           | McDonald & McDonald, 1973    |
| Churg et al, 1965        | Saccone & Coblenz, 1943      |
| Godwin, 1957             | Smart & Hinson, 1957         |
| Hochberg, 1951           | Wagner et al, 1972           |
| Hourihane, 1964          | Webster, 1965                |
| Mangnikan & Prior, 1963  | Whitwell & Rawcliffe, 1971   |
| McCaughey, 1958          | Winslow & Taylor, 1960       |
| McCaughey, 1965          |                              |
41. A high proportion of Mesotheliomata of pleura and peritoneum are associated with a history or other evidence of exposure to asbestos.
- |                   |                    |
|-------------------|--------------------|
| Ashcroft, 1968    | McEwen et al, 1972 |
| Bader et al, 1965 | McNulty, 1962      |



- |                               |                                 |
|-------------------------------|---------------------------------|
| Bohlig et al, 1970            | McVittie, 1965                  |
| Borow et al, 1967             | Newhouse, 1969                  |
| Borow et al, 1973             | Newhouse & Thompson, 1965a      |
| Castleman, 1974               | Newhouse & Thompson, 1965b      |
| Churg et al, 1965             | Newhouse & Wagner, 1969         |
| Cooper, 1967                  | Owen, 1951                      |
| Dalquen et al, 1969           | Owen, 1964                      |
| Editorial, Lancet, 1966       | Owen, 1965                      |
| Editorial, Lancet, 1973       | Pooley, 1973                    |
| Editorial, NEJM, 1965         | Selikoff et al, 1967            |
| Elmes, McCaughey & Wade, 1965 | Selikoff et al, 1964            |
| Elmes & Wade, 1965            | Selikoff et al 1965a            |
| Finlayson et al, 1971         | Selikoff et al 1965b            |
| Fletcher, 1972                | Selikoff & Hammond, 1968        |
| Fowler et al, 1964            | Selikoff, Hammond & Churg, 1972 |
| Godwin and Jagatic, 1970      | Sleggs et al, 1961              |
| Hagerstrand et al, 1968       | Sluis-Cremer, 1965              |
| Hammond et al, 1965           | Smith, 1952                     |
| Heard & Williams, 1961        | Stumphius & Meyer, 1968         |
| Hinson, 1965                  | Thomson, 1962                   |
| Hourihane, 1964               | Various, 1974                   |
| Hourihane, 1965               | Vigliani et al, 1965            |
| Jacob & Anspach, 1965         | Wagner, 1965                    |
| Keal, 1960                    | Wagner et al, 1971              |
| Kleinfeld et al, 1967         | Wagner et al, 1960              |
| Lieben & Pistawka, 1967       | Webster, 1973                   |
| Moigneteau et al, 1974        | Whitwell & Rawcliffe, 1971      |
| McCaughey et al, 1962         | Wood & Gloyne, 1934             |
| McDonald et al, 1970          | World Health Organization, 1973 |
| McDonald & McDonald 1973      | Wright, 1969                    |
| McDonald et al, 1971          |                                 |

42. Asbestosis is always evident in the lung of patients with Mesotheliomata.

- |                    |                            |
|--------------------|----------------------------|
| Fowler et al, 1964 | Whitwell & Rawcliffe, 1971 |
| Hourihane, 1964    |                            |

43. Cases of Mesothelioma without a history of exposure to asbestos do occur.

- |                         |                                 |
|-------------------------|---------------------------------|
| Lieben & Pistawka, 1967 | Webster, 1973                   |
| McEwen et al, 1972      | World Health Organization, 1973 |

44. Mesothelioma rarely develops until 15 to 20 years or more after exposure to asbestos, though cases occurring earlier have been reported.

- |                            |                            |
|----------------------------|----------------------------|
| Dalquen et al, 1969        | Owen, 1965                 |
| Hammond et al, 1965        | Selikoff et al, 1967       |
| Newhouse, 1969             | Selikoff et al, 1965a      |
| Newhouse & Thompson, 1965a | Wagner et al, 1971         |
| Owen, 1964                 | Whitwell & Rawcliffe, 1971 |





45. Increasing the severity and duration of exposure to asbestos both increase the risk of Mesothelioma.

Newhouse, 1969

46. Smoking does not seem to increase the danger of Mesothelioma.

Wagner, 1971

World Health Organization, 1973

Whitwell & Rawcliffe, 1971

47. The incidence of Mesothelioma is, so far as we can tell, increasing markedly.

48. Some have suggested that the increased use of asbestos over the last 20 years will lead to further increase in the incidence of Mesothelioma.

Stumphius & Meyer, 1968

Thomson et al, 1963

Thomson, 1965

Wright, 1969

49. At present, there is no evidence of increase in the incidence of Mesothelioma or other disease caused by asbestos in those not exposed occupationally or by propinquity to asbestos.

Enterline & Kendrick, 1967

Wright, 1969

World Health Organization, 1973

50. Not all asbestos is equally dangerous in causing Mesothelioma. Crocidolite is probably the most dangerous, with Chrysotile less so, but either can cause the tumour.

Harrington et al, 1961

Wagner & Berry, 1969

Pooley, 1973

Wagner et al, 1971

Timbrell et al, 1971

World Health Organization, 1973

Wagner, 1970

51. Geographic variation in the incidence of Mesothelioma in South Africa in those apparently with equal exposure has not been explained.

Harrington et al, 1971

Webster, 1973

Sluis-Cremer, 1965

Wright, 1969

Timbrell et al, 1971

52. Instillation of asbestos into the pleural cavity can cause Mesothelioma and other tumours in various species of animal, but not all reports of such instillation record the induction of tumours. Some record only Fibrosis.

Berry & Wagner, 1969 - rat - tumours

Davis, 1974 - rat, mice, guinea pigs - fibrosis

Davis & Coniam, 1973 - mice

Gloyne, 1930 - rabbit - fibrosis

Gross & Harley, 1973 - rat - tumours

Hunter & Thomson, 1973 - rat - tumours



McLachlan & Wagner, 1974 - rat - tumours  
 Peacock & Peacock, 1965 - fowl - tumours  
 Selikoff et al, 1967 - review  
 Smith, Miller, Churg et al, 1965 - hamster - tumours  
 Smith, Miller, Elasser et al, 1965 - hamster - tumours  
 Stanton & Wrench, 1972 - rat - tumours  
 Stanton, 1974  
 Vorwald et al, 1951 - guinea pig, rat, rabbit - fibrosis  
 Wagner, 1962 - rat - tumours  
 Wagner, 1970 - rat - tumours  
 Wagner, 1972 - rat - tumours  
 Wagner & Berry, 1969 - rat - tumours

53. An increased incidence of Bronchogenic Carcinoma in those with asbestosis became evident in 1947, though earlier reports had noted coincidence of the two lesions. As many as 50% of men with Asbestosis have been reported to develop carcinoma.

Anon, 1960	Holleb & Angrist, 1942
Bader et al, 1965	Homberger, 1943
Bonser et al, 1955	Isselbacher et al, 1953
Borow et al, 1967	Lieben, 1966
Buchanan, 1965	Lynch & Cannon, 1948
Chauvet, 1958	Lynch & Smith, 1935
Cooper, 1967	Mancuso & Coulter, 1963
Cordova et al, 1962	Moigneteau et al, 1974
Cureton, 1948	McCullagh, 1974
Desmoules et al, 1941	Newhouse & Wagner, 1969
Doll, 1955	O'Donnell & Mann, 1957
Editorial, Lancet, 1973	Smith, 1952
Gloyne, 1935	Stoll et al, 1951
Gloyne, 1936	Telischki & Rubenstone, 1961
Heard & Williams, 1961	

54. Many other reports detail a high incidence of Bronchogenic Carcinoma in asbestos workers without stating whether or not fibrosis was present.

Borow, 1973	Keal, 1960
Cooper, 1967	Kleinfeld et al, 1967
Doll, 1964	Knox et al, 1965
Dunn & Weir, 1965	Knox et al, 1968
Editorial, Lancet, 1966	McDonald et al, 1971
Editorial, NEJM, 1965	McVittie, 1965
Elmes & Wade, 1965	Newhouse, 1969
Enterline, 1965	Newhouse & Berry, 1973
Enterline et al, 1972	Newhouse et al, 1972
Enterline, DeCoulfe, et al, 1973	Selikoff et al, 1967
Enterline & Kendrick, 1967	Selikoff et al, 1964
Fletcher, 1972	Selikoff et al, 1965a
Gloyne, 1951	Selikoff, Hammond & Churg, 1968
Hill et al, 1966	Smither, 1965
Hinson, 1965	Vigliani, 1965
Hueper, 1951	Wagner et al, 1971
Hueper, 1955a, b	World Health Organization, 1973
Hueper, 1965	



55. Rarely, it has been reported that there is no excess of lung cancer in asbestos workers.
- Braun and Truan, 1958 (Quebec)      Hourihane et al, 1966 (London)  
Cartier, 1955 (Quebec)
56. Reports in the last several years have shown in addition increase in the incidence of Bronchogenic Carcinoma in men exposed to asbestos but without pulmonary fibrosis, or carcinoma in a much greater number of men than develop fibrosis.
- Enterline & Henderson, 1973      Knox et al, 1968  
Hammond et al, 1965      Selikoff & Hammond, 1968  
Jacob & Anspach, 1965
57. Carcinoma of the lung rarely develops in asbestos workers until 15 - 20 years or more from first exposure.
- Hammond et al, 1965      Selikoff et al, 1965a  
Newhouse, 1969      Selikoff & Hammond, 1968  
Selikoff et al, 1967
58. Carcinoma of the lung is more likely in those heavily exposed to asbestos, but may occur in those only slightly exposed, though some reports have found no excess in those only lightly exposed. Duration of exposure is also important.
- Berrey et al, 1972      Newhouse, 1969  
Enterline et al, 1972      Newhouse et al, 1972  
Enterline, De Coufle et al, 1973      Selikoff et al, 1967  
Enterline & Kendrick, 1967      Wagner et al, 1971  
Knox et al, 1965      World Health Organization, 1973  
Knox et al, 1968      Wright, 1969  
Lieben, 1966
59. Reducing dust exposure has reduced the incidence of carcinoma of the lung to near that in the general population.
- Editorial, Lancet, 1966      Lieben, 1966  
Knox et al, 1965      Wagner et al, 1971  
Knox et al, 1968
60. The risk of carcinoma of the lung in asbestos workers is greater in those who smoke.
- Berry et al, 1972      Selikoff, Hammond & Churg, 1968  
Churg & Kannerstein, 1970      Wagner et al, 1971  
McCullagh, 1974      World Health Organization, 1973  
Newhouse et al, 1972      Wright, 1969  
Selikoff & Hammond, 1968
61. No special type of Bronchogenic Carcinoma is associated with asbestos.
- Chauvet, 1958  
Kannerstein & Churg, 1972





62. All types of asbestos may cause carcinoma of the lung.  
World Health Organization, 1973  
Wright, 1969
63. Attempts to quantitate the risk of carcinoma of the lung by multiplying a factor indicating intensity of exposure by its duration do not seem well founded.  
Ashcroft, 1968a  
Cauna, 1965  
Cooper, 1965  
Glover, 1973
64. Experimentally, inhalation of asbestos in animals may lead to the development of tumours in the lung or mesotheliomata in the pleura, or may enhance the effect of carcinogens.  
Gross et al, 1967 (rat)  
Lynch et al, 1957 (mice)  
Miller et al, 1965  
Smith et al, 1970 (hamster)  
Wright, 1969 (review)
65. Exposure to asbestos is associated with an increased incidence of carcinoma of the stomach and colon.  
Bader et al, 1965  
Bonser et al, 1955  
Borow et al, 1973  
Enterline et al, 1965  
Enterline et al, 1972  
Enterline & Kindrick, 1967  
Enticknap & Smither, 1964  
Hammond et al, 1965  
Kleinfeld et al, 1967  
Newhouse, 1969  
Newhouse et al, 1972  
Selikoff et al, 1967  
Selikoff et al, 1964  
Selikoff et al, 1965a  
Selikoff & Hammond, 1968  
Wagner et al, 1971  
World Health Organization, 1973
66. Asbestos fibres are present in drinking water, and in many other potables.  
Biles & Emerson, 1968 (English beer)  
Cunningham & Pontefract, 1971 (Canadian & U.S. beer, wines, soft drinks, water)  
Cunningham & Pontefract, 1973 (ditto)  
Dourmashkin & Dougherty, 1961 (culture media)  
Kay, 1974 (water)  
Olson, 1974 (water)
67. There is no evidence that asbestos fibres in water and potables are dangerous.  
World Health Organization, 1973
68. Asbestos can be demonstrated in the spleen, abdominal lymph nodes, and other tissues.  
Godwin & Jagatic, 1970  
Keal, 1960



69. Asbestos fibres given experimentally into the rat stomach, or fed to rats, can be recovered from the omentum, spleen, liver, kidney, lung, brain and blood, and from the wall of the gut.
- Cunningham & Pontefract, 1973      Westlake et al, 1965  
Pontefract & Cunningham, 1973
70. No association between asbestos and carcinoma of the ovary has been established.
- Keal, 1960      World Health Organization, 1973  
Wagner et al, 1971
71. Reports from the United Kingdom suggest a relationship between exposure to asbestos and carcinoma of the larynx, but this suggestion remains to be confirmed, and has been questioned.
- Holmes, 1973      Stell & McGill, 1973  
Newhouse & Berry, 1973
72. A relationship between asbestos and haemopoietic and other tumours has been suggested, but not established.
- Gerber, 1970
73. Early reports questioned an increased risk of tuberculosis in those with asbestosis, but this has not been confirmed.
- Bonser et al, 1955      Lynch & Cannon, 1948  
Ellman, 1933      Smither, 1965  
Enterline, 1965      Vorwald et al, 1951  
Gardner & Cummings, 1931      Wood & Gloyne, 1930  
Gloyne, 1951      Wood & Gloyne, 1931  
Lanza et al, 1935      Wood & Gloyne, 1932
74. From the foregoing, it seems clear:
- A) Occupational exposure to asbestos can cause pulmonary fibrosis
  - B) Occupational exposure to asbestos can cause pleural plaques
  - C) Occupational exposure to asbestos can cause Mesothelioma of the pleura or peritoneum
  - D) Patients with Asbestosis have an increased risk of developing carcinoma of the lung
  - E) Occupational exposure to asbestos does increase the risk of developing carcinoma of the lung in those who do not develop pulmonary fibrosis.
  - F) Occupational exposure to asbestos is probably associated with an increased incidence of carcinoma of the stomach and colon
  - G) Occupational exposure to asbestos may be associated with an increased risk of carcinoma of the larynx
75. The problem is to decide fairly what should be the criteria for compensating those so exposed who develop one of these conditions.



76. It seems clear that all those with occupational exposure to asbestos of sufficient severity, who develop pulmonary fibrosis of the type recognized as caused by asbestos, 10 or more years after the beginning of exposure, and who suffer disability from the fibrosis, should be compensated, as has been customary in the past.
77. Those who have occupational exposure to asbestos and develop only pleural plaques without disability should not be compensated.
78. All those with occupational exposure to asbestos who develop Mesothelioma 15 or more years after the initial exposure should be compensated, as has been customary.
79. All those who have Asbestosis, in the sense of pulmonary fibrosis, who develop carcinoma of the lung should be compensated, as in the past.
80. The major problem is those who develop carcinoma of the lung, who have been exposed to asbestos occupationally, but have no evidence of pulmonary fibrosis. Some at least of these men should be compensated. Perhaps a good rule would require
  - A) A history of occupational exposure
  - B) An interval of 15 years between start of exposure and development of carcinoma
81. No easy rule is available to determine the duration and intensity of exposure to asbestos needed to make an asbestos worker eligible for compensation if he develops carcinoma of the lung. All the population of Ontario has been exposed to asbestos and can be assumed to have asbestos in their lung. Where then should be the point at which occupational exposure is such as to justify compensation? No data exist to indicate the answer to this question. All that can be suggested at this time is that each case be considered separately, and reasonable exposure be accepted. No standard based on fibre count seems justified.
82. Similarly, some latitude in requiring a 15 year interval from first exposure before allowing compensation for carcinoma of the lung should also be given. Very high levels of exposure could justify an award in a patient with a shorter latent period.
83. At this time, the evidence seems insufficient to justify compensation for an asbestos worker who develops carcinoma of the stomach, colon or larynx. However, evidence in this matter is accumulating, and each case should be evaluated in the light of the evidence as it develops.
84. Studies to evaluate the intensity of contamination of the lung with asbestos have been begun at the Toronto General Hospital. The concentration of asbestos fibres in the lung will be determined to see if we can devise a standard which would indicate those unusually heavily exposed. Routine studies will be by light microscopy, with the use of the electron microscope in selected cases.





85. Studies could also be initiated to determine if there is a real association between exposure to asbestos and carcinoma of the larynx. In Ontario a high proportion of carcinoma of the larynx are treated at the Princess Margaret Hospital, and most of the rest must be treated at other radiation centres, so that conditions make a study easy. Those treated surgically could probably be traced by the E.N.T. Surgical Group, but even if not most cases do come to radiotherapy.

A prospective or retrospective study could easily be mounted, and could well determine the reality of the suggested association, and its strength.

If this were done, the investigation should be well planned by a skilled epidemiologist, together with an otolaryngologist, a radiotherapist and a pathologist. I would be glad to assist in any such endeavour.

Probably Selikoff's group should not be consulted, as their position is already in large part known, and Professor Morgan has perhaps weakened his position by his unfortunate study using patients from the Toronto General Hospital. If Doll could be interested, that would be ideal.



ASBESTOS IN THE LUNG

TABLE A

<u>AUTHOR</u>	<u>METHOD</u>	<u>PLACE</u>	<u>FREQUENCY %</u>
Anjilvel et al, 1966	Smear	Montreal (Miami)	48 28)
Ashcroft, 1968a	Smear	Tyneside	20
Bignon et al, 1970	Digestion	France	100
Cauna et al, 1965	Smear	Pittsburgh	41
Editorial, JAMA, 1966		Miami	30 (male) 20 (female)
Elmes, McCaughey & Wade, 1965a	Section	Belfast	14-27 (male)
Hagerstrand et al, 1968	Section	Malmö	35
Hagerstrand et al, 1973	Section	Malmö	48
Langer, Bader et al, 1971	Em	New York	48
Langer, Selikoff et al, 1971	Em	New York (London)	86 86)
Meurman, 1966	Section	Finland	57
McPherson et al, 1969	Section	Glasgow	44
Penman et al, 1970	Section	Dunedin	3
	Digestion	Dunedin	86
Polliack et al, 1968	Smear	Jerusalem	26
Roberts, 1967	Smear	Glasgow	23
Rosen et al, 1972	Digestion	New York	90
Selikoff & Hammond, 1968	?	New York	30-60
Selikoff & Nicholson et al, 1972	?	New York	3-55
Smith & Naylor, 1972	Digestion	Ann Arbor	100
Thomson, 1965	Smear	Capetown	30 (male)
Thomson & Graves, 1966		Miami	20 (female)
Thomson et al, 1963	Smear	Cape Town	26
Utidge et al, 1968	Digestion	Pittsburgh	97
Warnock et al, 1975	Digestion	Chicago	100
Xipell et al, 1969	Digestion	Melbourne	44



## ASBESTOS DISEASE - SUPPLEMENTARY REPORT, APRIL 15, 1976

DR. A. C. RICHIE, PROFESSOR OF PATHOLOGY, UNIVERSITY OF TORONTO

- A1. This report considers particularly the relationship between exposure to asbestos and the development of carcinoma in the alimentary tract.
- A2. This report should be read in conjunction with my earlier interim report on asbestos disease of 1975.
- A3. Recent work had done little to modify the conclusions set out in that report except as noted hereunder.

Comments on Earlier (1975) Report

- A4. There is increasing evidence to suggest that asbestos powder and perhaps very short asbestos fibres have little fibrogenic or carcinogenic power in animals, though a firm conclusion is not yet justified.

Gross, 1974  
Stanton, 1974

Hardy, 1975

- A5. Addition of croton oil, benzpyrene, or heating may increase the carcinogenicity of asbestos in animals, and infection with *Candida albicans* increased its fibrogenicity. Adding iron oxide had no effect and asbestos reduced the carcinogenicity of  $\text{PuO}_2$  in rat lung.

Jagatic, Rubnitz, Godwin, Weiskopf, 1967  
Roe, Walters, Harrington, 1966  
Committee on Biological Effects, 1971

Sanders, 1975  
Shabad, Pylev, et al, 1974  
Zaidi, Shankar, Dogra, 1973

- A6. Electron microscopy shows that the lung often contains small uncoated asbestos fibres, often of chrysotile. These submicroscopic fibres are more numerous than asbestos bodies visible by light microscopy. Only 10-30% of asbestos fibres are visible to light microscopy. The significance of these small fibres is unknown.

Ashcroft and Heppleston, 1973  
Langer, Baden, Hammond, Selikoff, 1971  
Langer, Rubin, Selikoff, 1972  
Langer, Selikoff, Sastre, 1971  
Ashcroft, Heppleston, 1973

Gross, 1974  
Hardy, 1975  
Pooley, 1973  
Bignon, et al, 1970  
Davis, 1965

- A7. Asbestosis has been associated with an increase in rheumatoid factor, an increase anti-nuclear antibody in the blood, and a decrease in the response of T cells to phytohaemagglutinin and such agents.

Kang, Sera, Orochi, Yamamura, 1974  
Pernis, Vigiliani, Selikoff, 1965  
Rickards, and Barrett, 1958

Turner-Warwick, 1973  
Turner-Warwick, Haslam, 1971  
Turner-Warwick, Parkes, 1970





- A8. Chrysotile and to a lesser degree other kinds of asbestos causes haemolysis in vitro.
- Harrington, Miller, Macnab, 1971                      Dolgner, 1969  
Schnitzer, Pundsack, 1970                      Szentei, 1969  
Gabor, Anca, 1975
- A9. The frequency of the HL-A system gene W 27 is increased in patients with asbestosis.
- Mercant, Klouda, Soutar, et al, 1975
- A10. Asbestos may induce chromosomal changes in cells grown in culture.
- Sincock, Seabright, 1975
- A11. The incidence of asbestosis may be falling though asbestos-related cancer, pleural plaques and pleural effusions are increasing.
- Brouet, Bignon, Bonnaud, Goni, 1971
- A12. Asbestos may damage mesothelial cells and fibrocytes as well as macrophages.
- Divertie, Cassan, Brown, 1975                      Davis, 1974a  
Richards, Hext, Blundell, et al, 1974                      Davis, 1974b
- A13. Serum protects macrophages against injury by asbestos, and the protein covering of asbestos fibres may do so as well.
- Sakabe, Koshi, Hayaski, 1971                      Allison, 1971
- A14. Calcified plaques may be found on the liver in asbestosis.
- Fondimare, Duwood, et al, 1974.
- A15. Mesothelioma can occur in people who have only moderate exposure to asbestos and who show no other evidence of asbestos injury.
- Ashcroft, 1973
- A16. Mesothelioma may occur with increased frequency in people who live near an asbestos plant.
- Hain, Dalquen, et al, 1974                      Greenberg, Davies, 1974
- A17. Particular note might be taken of a recent Finnish report that the risk of developing carcinoma of the lung is increased 1.4 times in non-smoking asbestos workers, 12 times in smokers not exposed to asbestos, and 17 times in asbestos workers who smoke. In New York in 1968, it was estimated that asbestos workers who smoke increase 92 times the risk of dying of carcinoma of the lung. Other reports concurring in stressing the great importance of smoking in the causation of carcinoma of the lungs in asbestos workers are listed in section 60 of the original report.
- Meurman, Kiviluoto, Hakamam, 1974                      Selikoff, Hammond, Churg, 1968



A18. Gerbils inhaling asbestos fibres developed alveolar proteinosis.

Reeves, Puro, Smith, 1974

A19. Additional references to other sections of the earlier interim report are listed in appendix A6.

#### Asbestos and Alimentary Carcinoma

- A20. Table A1 lists reports on the relationship between exposure to asbestos and carcinoma of the alimentary tract. It should be noted that the successive papers by Selikoff and his colleagues Bader, Hammond, and Borow refer in part at least to the same group of patients. The figure "increase" records the excess in alimentary carcinoma observed in asbestos workers as compared with the control used for comparison.
- A21. Many of the authorities quoted in Table A1 also record the frequency of carcinoma of the lung in asbestos workers. For comparison, these figures are given in Table A2.
- A22. The figures in Table A2 are congruent with those of the other authorities listed in sections 53, 54, 56 and 58 of my earlier report.
- A23. When the figures in Table A1 are examined there seems little doubt that the risk of developing a carcinoma of the alimentary system is increased in asbestos workers. In asbestos workers, the risk of developing carcinoma of the alimentary system is about twice that in controls.
- A24. In comparison, the figures in Table B2 show that the risk that an asbestos worker will develop carcinoma of the lung is increased by 5 or 6 times at least.
- A25. A considerable weakness in the figures quoted in Table A1 is that most come from Selikoff and his colleagues. Their work is of a high order, and their conclusions most acceptable, but more confirmation from other sources would be reassuring. Selikoff's study of 17800 insulation workers is very impressive, and one of the very few in which a statistical analysis seems reliable.
- A26. In the esophagus, only Selikoff's study of 17800 asbestos workers shows an increased risk of carcinoma of 2.8 times, significant at the 0.001 level, though Selikoff's finding of an increased incidence of oesophageal and gastric carcinoma considered together in 632 insulation workers, and the similar report by McDonald and his colleagues concur.
- A27. The evidence suggesting that carcinoma of the stomach is increased in asbestos workers is weaker. Selikoff's study of 17800 insulation workers shows only an increase of 1.6 times, significant only at the 0.05 level. McDonald and his colleagues found only a 1.4 times increase in those heavily exposed, as compared with those minimally exposed, which does not seem to be significant.



- A28. The increased risk of carcinoma of the colon and rectum in asbestos workers is plainer. Selikoff's study of 17800 insulation workers showed only an increase of 1.6 times, but it was highly significant, at the 0.001 level. His other studies and McDonald and his colleagues found a greater increase risk, of 3 to 4 times.
- A29. The studies in which gastro-intestinal carcinomata are grouped together further support the reality of the increase in carcinoma of the colon and rectum in asbestos workers. The majority of gastro-intestinal carcinomata are in the large bowel, 47 carcinomata of colon or rectum as compared with 18 gastric carcinomata in Selikoff's 17800 workers. With the marked exception of the Finnish authophyllite miners, most of the investigators found that the risk of gastrointestinal carcinoma was increased 2 to 3 times in Asbestos workers.
- A30. In contrast, Enterline's studies of asbestos workers strikingly fail to support the theory that carcinoma of the digestive system is increased in asbestos workers, though his large studies do show the usual increase in carcinoma of the lung, also evident in Meurman's Finnish workers.
- A31. Selikoff's study of 17800 insulation workers shows that no excess of alimentary carcinoma was evident until 20 years after the beginning of exposure.
- Selikoff, 1976
- A32. In a letter, Dr. Selikoff comments that there is no association between smoking and carcinoma of the stomach, colon or rectum, but the increase in carcinoma of the oesophagus is found only in smokers.
- A33. If asbestos does increase the frequency of gastrointestinal carcinoma, asbestos bodies should be more numerous in the lungs of patients with carcinoma of the colon. Table A3 shows studies of this sort. The findings are equivocal.
- A34. In a more direct attempt to show that asbestos caused carcinoma of the colon, Rosen and his colleagues found atypical asbestos fibres in 6 of 12 carcinomata of the colon and in 1 of 4 non-neoplastic conditions. The significance is not apparent. The bodies may not have been related to asbestos.
- Rosen, Savino, Melamed, 1974.
- A35. Asbestos bodies can penetrate the tissue, and may be present in hilar or mediastinal lymphnodes in the spleen, small bowel peritoneum, or thyroid in patients with mesothelioma or asbestosis.

Godwin, Jagatic, 1970

Keal, 1960





- A36. Experimentally, asbestos fibres ingested into the stomach in rats were found by electron microscopy in the blood, spleen, liver, kidney, omentum, muscle, lung and brain. In rats fed asbestos, fibres have been found by electron microscopy in the mucosa of the intestine or colon, in the mesentry, and the lung. Fibres were also found in the tissues in control rats.
- Gross, Harley, Swinburne, Davis, Greene, 1974  
Cunningham, Pontefract, 1973  
Pontefract, Cunningham, 1973  
Westlake, Spjut, Smith, 1965
- A37. In rats, asbestos given intravenously during pregnancy was found in the foetus, and in one rat asbestos given intrapleurally penetrated into the liver.
- Cunningham, Pontefract, 1974      Morgan, Holmes, Gold, 1971
- A38. As shown in Table A4 asbestos fibres can be found in many potable fluids, including drinking water.
- A39. There is no excess cancer mortality in Duluth or in other areas where the drinking water contains much asbestos, and the frequency of gastro-intestinal cancer has decreased, during the period in which use of asbestos cement pipe has increased.
- Gross, Harley, et al, 1974      Masson, McKay, Miller, 1974  
Olson, 1974
- A40. There is no reason to think that the presence of asbestos fibres in potable fluids brings any risk to health.
- World Health Organization, 1973      Gross, 1974 a  
Masson, McKay, Miller, 1974      Gross, 1974 b  
Gross, Harley, et al, 1974      Olson, 1974
- A41. Some writers doubt if asbestos has any relation to carcinoma of the alimentary tract.
- Gross, Harley, et al, 1974      Gross, 1974 a  
Gross, 1974 b
- A42. One paper wonders why carcinoma of the lung and stomach are not compensable in asbestos workers in France.
- Pariente, Guttieres, 1974
- A43. In conclusion, the evidence available on the relationship between exposure to asbestos and carcinoma of the alimentary system is still inadequate to allow a final conclusion, but there is now strong reason to believe:
- a) that the incidence of carcinoma of the colon and rectum is increased in asbestos workers;



- b) that an asbestos worker is about twice as likely to develop carcinoma of the colon and rectum as is a man not exposed to asbestos;
- c) the incidence of carcinoma of the stomach is probably increased in asbestos workers, though the evidence is weaker than for the colon;
- d) an asbestos worker might have 1.5 to 2 times a greater risk of developing carcinoma of the stomach than the general population;
- e) the incidence of carcinoma of the oesophagus is probably increased in asbestos workers, but his conclusion rests almost entirely on Selikoff's recent study, and needs confirmation from other sources;
- f) if there is an increase in the incidence of carcinoma of the oesophagus in asbestos workers, the risk is probably increased by about two times;
- g) in short, it would be reasonable to conclude that on the evidence now available, carcinoma of the alimentary tract is increased in frequency in asbestos workers, and that an asbestos worker is about twice as likely to develop such a carcinoma as is the population at large;
- h) presumably the increase in carcinoma of the alimentary system is caused by asbestos fibres swallowed, but there is no evidence on this point.

A44. If this conclusion is correct, and if the figures for carcinoma of the lung given by Meurman and his colleagues are correct, an asbestos worker who does not smoke runs twice the usual risk of developing an alimentary carcinoma, but only 1.4 times the usual risk of developing a carcinoma of the lung, while an asbestos worker who smokes has twice the normal risk of carcinoma of the digestive system, but 17 times the usual risk of carcinoma of the lung.

Meurman, Kivilvoto, Hakama, 1974

A45. It has been suggested that the high incidence of carcinoma of the stomach in Japanese might be due to asbestos in the talc used to dust Japanese rice.

Merliss, 1974

#### Asbestos and Other Cancers

A46. Table A5 summarizes reports suggesting that carcinoma of the larynx occurs with increased frequency in asbestos workers.



A47. The association of asbestos exposure with carcinoma of the lung rests largely on the reports of Stell and McGill from Liverpool, though the concurrent report by Shettigara from Toronto should be noted.

A48. Objection to Stell and McGill's work has been voiced, and it is striking that no confirmation comes from Selikoff's detailed studies.

Holmes, 1973

A49. Those cases in which carcinoma of the larynx is associated with asbestos have usually shown a latent period of over 20 years, though in some it was under 5 years.

Stell, McGill, 1973 a

Stell, McGill, 1973 b

Newhouse, Berry, 1973

Stell, McGill, 1975

Shettigara, Morgan, 1975

A50. Most of those in whom carcinoma of the larynx is associated with asbestos have been smokers.

Libshitz, Wershba, et al, 1974

Stell, McGill, 1973 a

Stell, McGill, 1973 b

Stell, McGill, 1975

Newhouse, Berry, 1973

Shettigara, Morgan, 1975

A51. The best conclusion at this time is that the reality of the association between exposure to asbestos and carcinoma of the larynx remains to be established, though there is strong reason to investigate this possibility further.

A52. An increased frequency of asbestos bodies in the lungs of patients with carcinoma of the breast is reported in one series.

Doniach, Swettenham, et al, 1975

### Conclusion

A53. To restate and expand the conclusions of my earlier (1975) report:

A. Exposure to asbestos can cause pulmonary fibrosis.

B. Exposure to asbestos can cause pleural plaques.

C. Exposure to asbestos can cause pleural effusions.

D. Exposure to asbestos can cause mesothelioma of the pleura or peritoneum.

E. The great majority of mesotheliomata are due to exposure to asbestos.

F. Patients with asbestosis have a greatly increased risk of developing carcinoma of the lung.





- G. Exposure to asbestos increases the risk of developing carcinoma of the lung even if no pulmonary fibrosis is induced.
  - H. The risk of carcinoma of the lung is very high in asbestos workers who smoke.
  - I. The risk of carcinoma of the lung is increased in asbestos workers who do not smoke, but the increase is slight.
  - J. Exposure to asbestos is associated with an increased risk of developing carcinoma of the colon and rectum.
  - K. Exposure to asbestos is probably associated with an increased risk of carcinoma of the stomach and oesophagus.
  - L. Exposure to asbestos may increase the risk of carcinoma of the larynx, but the association remains to be proved.
  - M. There is no evidence that carcinoma of other sites or other kinds of cancer are unduly frequent in asbestos workers.
- A54. From the foregoing, and the data in this report and in my earlier report on disease caused by asbestos, it seems reasonable to suggest:
- A. that all with exposure to asbestos of reasonable severity who develop pulmonary fibrosis of the type recognized as caused by asbestos, 10 or more years after the beginning of exposure, and who suffer disability from the fibrosis, should be compensated;
  - B. that those exposed to asbestos who develop only pleural plaques, without disability, should not be compensated;
  - C. that all with any exposure to asbestos who develop mesothelioma should be compensated;
  - D. that all who have asbestosis, in the sense of pulmonary fibrosis, who develop carcinoma of the lung should be compensated.
  - E. that individual consideration be given to all with exposure to asbestos who develop carcinoma of the lung but do not have pulmonary fibrosis;
  - F. that those with exposure to asbestos who develop carcinoma of the lung should usually be compensated if: -
    - a) there is a history of adequate exposure to asbestos, or clear histological evidence of such exposure;
    - b) 15 or more years have elapsed since the beginning of exposure to asbestos.
  - G. that in special circumstances, such as unusually massive exposure to asbestos, some who developed carcinoma of the lung



in less than 15 years after the beginning of exposure to asbestos should be compensated, bearing in mind that the susceptibility to injury by asbestos probably varies from person to person, and that there are no reliable criteria for determining either the minimal intensity of exposure needed to cause carcinoma, or the minimum time necessary for its induction;

- H. that no consideration be given to whether or not the claimant smokes in awarding compensation for carcinoma of the lung in those exposed to asbestos. Smoking very greatly increases the risk of developing carcinoma of the lung in those exposed to asbestos, but men now or previously employed in asbestos plants could not have known that this was the case;
- I. that consideration be given to warning all working with asbestos of the very great danger they run of carcinoma of the lung if they smoke;
- J. that consideration be given to compensating those exposed to asbestos who develop carcinoma of the colon or rectum;
- K. that provisionally, and with full realization that our knowledge is incomplete, that revision of these criteria will probably prove necessary within a few years, and that half of those exposed to asbestos who develop carcinoma of the colon or rectum would have developed the cancer even if never exposed to asbestos, all exposed to asbestos who develop carcinoma of the colon or rectum should be compensated if: -
  - a) there is clear evidence of considerable exposure to asbestos;
  - b) not less than 20 years have elapsed since the beginning of exposure.

No clear definition of what is meant by "considerable" in (a) above is possible at this time.

- L. that consideration be given to compensating those exposed to asbestos who develop carcinoma of the stomach or oesophagus, realizing that the evidence associating these cancers with exposure to asbestos is weaker than for carcinoma of the colon and rectum.
- M. that provisionally, as for carcinoma of the colon and rectum, those exposed to asbestos who develop carcinoma of the stomach or oesophagus should be compensated if: -
  - a) there is clear evidence of considerable exposure to asbestos;
  - b) not less than 20 years have elapsed since the beginning of exposure.

No clear cut definition of what is meant by "considerable" in (a) above is possible at this time.



- N. that no compensation be given those exposed to asbestos who developed carcinoma of the larynx, but that the possible relationship between carcinoma of the larynx and asbestos be kept under review.
- O. that any decisions taken be reviewed in 3 years within light of any new evidence then available.





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Table A1

Relation of Asbestos Exposure to  
Carcinoma of the Alimentary Tract

<u>Oesophagus</u>	Author	Patients	Finding	Increase
<u>Stomach</u>	Selikoff, 1976	17800 Insulation Workers	14 Observed, 5.4 expected	2.6 <sup>1</sup>
	Bader, et al, 1965	Asbestos Workers	2 of 17 developed carcinoma	-
	Selikoff, et al, 1968	Insulation Workers	3 in 370, 0.6 expected	5.0
	McDonald, et al, 1971	Asbestos Workers	1.4% death rate carcinoma stomach and oesophagus with heavy exposure, 1.0% with light	1.4 15
<u>Colon and Rectum</u>	Selikoff, 1976	17800 Insulation Workers	18 observed, 11.2 expected	1.6 <sup>2</sup>
	Selikoff, 1976	632 Insulation Workers	20 carcinoma of stomach and oesophagus observed, 6.5 expected	3.1
	Selikoff, et al, 1968	Insulation Workers	5 in 370 exposed, 1.2 expected	4.2
	Dohner, et al, 1975		2 cases with carcinoma of lung and carcinoma of colon	



Table A1

Colon and Rectum Cont'd

Author	Patients	Finding	Increase
McDonald, et al, 1971	Asbestos Workers	0.8% death rate with heavy exposure, 0.3% with light	2.7
Selikoff, 1976	17800 Insulation Workers	47 Observed, 28.6 expected	1.6 <sup>1</sup>
Selikoff, 1976	632 Insulation Workers	23 Observed, 7.6 expected	3.0
Selikoff, et al, 1964	Insulation Workers	29 carcinomas found, 9.4 expected	3.1
Selikoff, et al, 1965	Insulation Workers	3 times expected	3.0
Hammond, et al, 1965	Insulation Workers	29 carcinomas found, 9.4 expected	3.1
Jacob, et al, 1965	Asbestos Workers	4 in 102 with asbestosis, (22 had carcinoma of the lung)	-
Kleinfeld, et al, 1967	Asbestos Insulators	15.2% of deaths, 3.9 expected	3.9
Selikoff, et al, 1967	Insulation Workers	May be increased	-
Wagner, et al, 1971	Asbestos Workers	"Evidence inconclusive"	-
Elmes, et al, 1971	Insulation Workers	17 "other carcinoma" in 170 workers, 5 expected, 11/17 from Gastro-intestinal tract	2.2

Gastro-Intestinal

- 16 -





Table A1

Gastro-intestinal Cont'd

Author	Patients	Finding	Increase
Borow, et al, 1973	Asbestos Workers	12/90 patients admitted to hospital	--
W.H.O., 1973		Some occupational groups have shown a slight increase.	-
Meurman, et al, 1974	Anthophyllite miners	2 Observed, 2.1 expected	1.0
Selikoff, 1976	611 Asbestos Workers	11 Observed, 6.3 expected	1.7
Selikoff, 1976	933 Amorite Workers	24 Observed, 12.5 expected	1.9
Enterline, 1965	Asbestos Workers	21 Cancers observed, 13.9 expected	1.5
Enterline, et al, 1967	Asbestos Workers:- building products friction products textiles	88.8 Observed, 96.3 expected 119.2 Observed, 96.3 expected 146.3 Observed, 96.3 expected	0.9 1.2 1.5
Enterline, et al, 1972	Asbestos Workers	59 Observed in 1464 workers, 45.9 expected	1.3
Enterline, et al, 1973 a & b	Asbestos Workers	No significant increase in 1348 workers	-

Digestive System

- 
- 1. Significance 0.001
  - 2. Significance 0.05



Table A2

Relation of Asbestos Exposure to  
Carcinoma of the Lung

Author	Patient	Finding	Increase
Enterline, 1965	Asbestos Workers	24 Observed, 11.9 expected	2.0
Selikoff, et al, 1964	Insulation Workers	45 Observed, 6.6 expected	6.8
Selikoff, et al, 1965	Insulation Workers	7 times expected	7.0
Hammond, et al, 1965	Insulation Workers	45 Observed, 6.6 expected	6.8
Bader, et al, 1965	Asbestos Workers	3 of 17 developed carcinoma	-
Enterline, et al, 1967	Asbestos Workers: - building products friction products textiles	130.4 Observed, 31.1 expected 123.7 Observed, 31.1 expected 228.6 Observed, 31.1 expected	4.2 4.0 7.4
Kleinfeld, et al, 1967	Asbestos Insulators	12 deaths in 46, 9 times expected. 26.1% of deaths, 3.1 expected	8.4
Selikoff, et al, 1967	Insulation Workers	1 in 5 will develop carcinoma of lung, 6 - 10 times expected	6.0 - 10.0
Selikoff, et al, 1968	Insulation Workers	24 in 370, 3.3 expected	7.3
Elmes, et al, 1971	Insulation Workers	17 times expected, 24 in 170 workers	17.0
McDonald, et al, 1971	Asbestos Workers	2.4% death rate with heavy exposure, 0.8% with light	3.0



Table A2 - Lungs Cont'd

Author	Patient	Finding	Increase
Enterline, et al, 1972	Asbestos Workers	59 Observed in 1464 workers, 23.7 expected	2.5
Borow, et al, 1973	Asbestos Workers	14 in 90 patients admitted to hospital	
Enterline, et al, 1973	Asbestos Workers	58 Observed in 1348 workers, 5.3 times expected	5.3
Meurman, et al, 1974	Anthophyllite miners	8 Observed, 2.4 expected	3.3
Selikoff, 1976	17800 Insulation Workers	753 Observed, 79.8 expected	4.7 <sup>1</sup>
Selikoff, 1976	611 Asbestos Workers	30 Observed, 10.2 expected	2.9
Selikoff, 1976	933 Amorite Workers	83 Observed, 12.5 expected	6.6
Selikoff, 1976	632 Insulation Workers	89 Observed, 12.2 expected	7.3





Table A3

Asbestos Bodies in the Lungs  
of Patients with Cancer

Author	Patients	Finding
Ashcroft, 1968	Routine autopsies	No increase in GI or lung cancer
Doniach, et al, 1975	Routine autopsies	In 70% with C <sub>a</sub> stomach, 45.4% expected In 46% with C <sub>a</sub> breast, 32% expected.
Hagerstrand, et al, 1968; Pooley, 1973	Autopsies	Asbestos bodies much more numerous in patients with mesothelioma than in controls
Utiddjian, et al, 1968	Routine autopsies	No relation to cancer
Warnock, et al, 1975 a & b	Routine autopsies	27% with lung carcinoma had over 50 per gram. 5% in controls



Table A4

Asbestos Fibres in Potable Fluids, etc.

Dourmashkin, et al, 1961	Tissue culture media	
Biles, et al, 1968	English beer	5000 per pint
Cunningham, et al, 1971	Canadian beer	4-6 x 10 <sup>6</sup> per litre
	U.S. beer	1-2 x 10 <sup>6</sup> per litre
	Sherry, Spanish, S.African, Canadian	2-4 x 10 <sup>6</sup> per litre
	Vermouth, French	2 x 10 <sup>6</sup> per litre
	Vermouth, Italian	12 x 10 <sup>6</sup> per litre
	Ginger Ale	12 x 10 <sup>6</sup> per litre
	Tonic water	2 x 10 <sup>6</sup> per litre
	Orange drink	2 x 10 <sup>6</sup> per litre
	Tap water - Ottawa, Toronto, Montreal, Drummondville, Asbestos Beauport, P.Q. Thetford Mines	3 x 10 <sup>6</sup> per litre
Kay, 1974	Snow - Ottawa	2-4 x 10 <sup>6</sup> per litre
		8-9 x 10 <sup>6</sup> per litre
		173 x 10 <sup>6</sup> per litre
		34 x 10 <sup>6</sup> per litre
Wehman, et al, 1974	Water in Ontario: - with filtration plant (15 towns) without filtration plant (7 towns)	0.1-2.8 x 10 <sup>6</sup> per litre 0.2-3.9 x 10 <sup>6</sup> per litre
	Gin	0-239 x 10 <sup>3</sup> per 10ml



Table A5  
Asbestos and Carcinoma of the Larynx

Author	Patients	Finding	Increase
Newhouse, et al, 1973	1327 Asbestos Workers under 2 years exposure over 2 years exposure	2 observed, 0.17 expected 0 observed, 0.02 expected	11.8 -
Stell, et al, 1973 a	100 cancer of head and neck	17 of 59 carcinoma larynx exposed (29%) 2 of 20 carcinoma pharynx exposed (10%) 2 of 11 carcinoma nose exposed (18%) 1 of 10 carcinoma hypopharynx exposed (10%) 2 of 100 controls exposed (2%)	
Stell, et al, 1973 b	100 carcinoma of larynx plus 100 controls	31 of 100 carcinoma of larynx exposed (31%) 3 of 100 controls exposed (3%)	
Libshitz, et al, 1974	2 cases	2 cases	
Stell, et al, 1975	119 carcinoma of larynx and controls	27.7% carcinoma of larynx exposed 2.5% of controls exposed	
Shettigara, et al, 1975	43 carcinoma of larynx and controls	10 of 43 carcinoma of larynx exposed (23%) 0 of 43 controls exposed (0%)	





ASBESTOS DISEASE - REPORT 3#, JULY 27, 1976

DR. A. C. RITCHIE, PROFESSOR OF PATHOLOGY, UNIVERSITY OF TORONTO

B1. This report is to clarify certain points which have arisen in discussion of my report on Asbestos Disease of April 15, 1976.

B2. There is no doubt that the inhalation of asbestos fibres can cause disease of the lung. This conclusion is supported by excellent studies by several groups of investigators in different parts of the world, and is generally accepted.

B3. There is general agreement that inhalation of asbestos fibres can cause five kinds of pulmonary disease, as set out in paragraph A 53, sections A to I, of my report of April 15, 1976.

B4. Those sections of paragraph A 53 state that the inhalation of asbestos fibres can cause:

1. pulmonary fibrosis
2. pleural plaques
3. pleural effusions
4. mesothelioma of the pleura or peritoneum
5. carcinoma of the lung

B5. There is also wide agreement, as set out in paragraph A 53, that:

1. the great majority of mesotheliomata are due to inhalation of asbestos fibres
2. the risk of developing carcinoma of the lung is greatly increased in those in whom the inhalation of asbestos causes pulmonary fibrosis
3. the risk of developing carcinoma of the lung is also increased in those who inhale considerable quantities of asbestos fibres but who do not develop pulmonary fibrosis
4. cigarette smoking greatly increases the risk that a person exposed to asbestos fibres will develop carcinoma of the lung.

B6. It also seems reasonable to conclude, though the evidence is less clear, that:

1. mesothelioma can develop in people who have only slight or moderate exposure to asbestos, and who show no other evidence of asbestos disease
2. the greater the contamination of the lungs with asbestos, the greater the risk of carcinoma of the lung
3. whatever the exposure to asbestos, only a small proportion of those exposed will develop either mesothelioma or carcinoma of the lung.



B7. To clarify further these matters, it is recommended that the studies to correlate the quantity of asbestos in the lung with the severity and type of pulmonary disease caused by asbestos be continued.

B8. In contrast to the general agreement that the inhalation of asbestos fibres increases the risk of developing a mesothelioma or a carcinoma of the lung, there is debate as to whether exposure to asbestos fibres increases the risk of developing other kinds of cancer, and in particular whether exposure to asbestos fibres increases the risk of developing carcinoma of the stomach or colon.

B9. The evidence available in the literature to support or refute the suggestion that the risk of developing carcinoma of the stomach or colon is increased in those exposed to asbestos fibres is set out in my report of April 15, 1976.

B10. My conclusions are set out in paragraph A 53, sections J to M.

B11. These conclusions are:

1. exposure to asbestos is associated with an increased risk of developing carcinoma of the colon or rectum
2. exposure to asbestos is probably associated with an increased risk of carcinoma of the stomach and esophagus
3. exposure to asbestos may increase the risk of carcinoma of the larynx, but the association remains to be proved
4. there is no evidence that carcinoma of other sites or other kinds of cancer are unduly frequent in asbestos workers.

B12. The conclusion that exposure to asbestos increases the risk of developing carcinoma of the colon or rectum, and probably increases the risk of developing carcinoma of the stomach and esophagus depends almost entirely on epidemiological data.

B13. These epidemiological studies are not as strong or as clear as those which link exposure to asbestos to lung disease.

B14. As emphasized in paragraph A25 of my report of April 15, 1976, the conclusion that exposure to asbestos increases the risk of developing gastrointestinal carcinoma rests very largely on the work of Selikoff and his colleagues, and in particular on their study of 17,800 insulation workers in the United States and Canada.

B15. The work of these investigators from the Mount Sinai Hospital in New York seems of a high order, and fully worthy of credence.

B16. Nevertheless, it would be highly desirable that their conclusion that exposure to asbestos increases the risk of developing gastrointestinal carcinoma be confirmed by another group of investigators and in another population.





B17. Table A1 of my report of April 15, 1976 summarizes the reports in the literature which consider the incidence of carcinoma of the alimentary tract in asbestos workers.

B18. If the reports by Selikoff and his colleagues are excluded, the other data listed in Table A1 suggest that carcinoma of the gastrointestinal tract may be increased in some asbestos workers, but fail to establish that this is the case.

B19. It should also be noted that to the best of my knowledge Selikoff's major study of 17,800 insulation workers in the United States and Canada has not been published. The only information I have about the findings in this study are the tables kindly sent to me by Dr. Selikoff earlier this year, and attached to my report of April 15, 1976 as Appendix 3 .

B20. I do not think that it would be proper to make these tables public without Dr. Selikoff's permission.

B21. There is little non-epidemiological evidence to support the suggestion that exposure to asbestos increases the risk of developing carcinoma of the gastrointestinal tract.

B22. Table A3 of my report of April 15, 1976 summarizes the few studies that have attempted to determine if the number of asbestos bodies in the lungs is increased in patients with cancer, as would probably be the case if exposure to asbestos did increase the risk of cancer. The findings are contradictory and inconclusive.

B23. Only one group of investigators have sought for asbestos fibres in gastrointestinal tumours. The significance of their findings is not apparent.

B24. Asbestos fibres have not been shown able to produce tumours of the gastrointestinal tract experimentally in animals. This may be because little attention has been given to this possibility, and proper experiments have not been undertaken.

B25. Asbestos fibres can be demonstrated in the intestine and in other extra-pulmonary tissues both in patients and in experimental animals.

B26. Thus my conclusion that exposure to asbestos fibres increases the risk of developing carcinoma of the colon and rectum and probably increases the risk of developing carcinoma of the stomach and esophagus rests almost entirely on epidemiological evidence, and principally on the work of Selikoff and his colleagues.

B27. This conclusion is my assessment of the evidence available. This evidence is not as complete as would be desirable, and further studies are needed to confirm that this conclusion is correct.

B28. Among the steps which could be taken to confirm or refute the conclusion that exposure to asbestos increases the risk of carcinoma of the colon and rectum, and probably increases the incidence of carcinoma of the stomach and esophagus, are:





1. the validity of the epidemiological data on which this conclusion rests should be assessed by an epidemiologist
2. if a suitable cohort of Ontario workers exposed to asbestos is available, the incidence of gastrointestinal carcinoma in this cohort should be established
3. any gastrointestinal carcinomata arising in people with significant exposure to asbestos should be studied to determine if they differ in any way from gastrointestinal carcinomata arising in people not so exposed
4. search should be made to determine if asbestos fibres are present in the colon adjacent to any gastrointestinal carcinomata that may arise in people significantly exposed to asbestos, and if so if they are more numerous than in the general population
5. the frequency of asbestos bodies in the lungs of patients with gastrointestinal carcinoma should be determined, to discover if these people are more heavily exposed to asbestos than is the population at large.

B29. It is usually assumed that if exposure to asbestos does increase the risk of developing gastrointestinal carcinoma the increased risk results from swallowing asbestos fibres, which penetrate the gastrointestinal mucosa, and so induce the carcinoma. There is no evidence that this is the case.

B30. As shown in Table A4 of my report of April 15, 1976, many potable fluids contain asbestos fibres. There is no evidence that drinking any of these fluids increases the risk of cancer, if the possible association between excessive consumption of alcohol and esophageal cancer is excluded.

B31. There is no excess of gastrointestinal or other cancer in Duluth and other areas in which the drinking water contains much asbestos.

B32. The widespread use of asbestos cement pipe has not caused any demonstrable increase in gastrointestinal cancer.

B33. Thus if there is an increase in gastrointestinal carcinoma in asbestos workers, this must be because they swallow much greater quantities of asbestos than are taken by drinking contaminated water, or because the asbestos to which the workers are exposed is in some way different.

B34. The suggestions set out in paragraph A 54 of my report of April 15, 1976 are based only on the medical evidence, and are intended only to serve as a basis for discussion.

B35. As my report of April 15, 1976 was prepared some time ago, I shall review the literature that has appeared since that time, to see if anything of relevance has appeared.



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ASBESTOS FIBRE DUST AND GASTRO-INTESTINAL MALIGNANCIES

## Review of Literature with Regard to a Cause/Effect Relationship.

A.B. Miller, B.A., M.B., B.Chir., M.R.C.P., F.R.C.P.(C), M.F.C.M.  
Director, Epidemiology Unit, National Cancer Institute of Canada.  
Professor, Dept. of Preventive Medicine at Biostatistics,  
University of Toronto.

1.0 INTRODUCTION

- 1.1 On July 23, 1976 I received a letter dated July 21 from Dr. W.J. McCracken, Executive Director, Rehabilitation Services Division of the Workmen's Compensation Board of Ontario, requesting me to carry-out a statistical and epidemiological evaluation of the world literature in order to facilitate a study of the cause/effect relationship between asbestos fibre dust effect and the development of gastro-intestinal malignancies, that the Workmen's Compensation Board had initiated. The Board had already received from Dr. A.C. Ritchie, a report on an evaluation of the literature from his standpoint as a specialist in pathology. Following a review of Dr. Ritchie's report, it had been concluded that "In so far as the world literature is concerned, it is apparent that this consists of epidemiological studies and statistical analyses, and it would further appear that a rather high percentage of the papers published leave much to be desired, as it relates to the correctness of the experiment as well as a severe failure to identify the specific malignancies and specific sites of malignancies viz. right versus left side of colon, lower 1/3 versus other areas of esophagus, pre-pyloric versus greater curve malignancies of stomach etc. Dr. McCracken had concluded that "We now require a critical assessment from a statistical and epidemiological evaluation relative to those papers which could and should be used and those which should be discarded as being of poor design."
- 1.2 On July 23, I wrote to Dr. McCracken indicating that I would be prepared to carry out the evaluation requested, and on August 3, I met with Dr. McCracken in the Offices of the Workmen's Compensation Board of Ontario, and discussed this further with him. At that meeting, I was given copies of many of the available papers, together with copies of Dr. Ritchie's reports.
- 1.3 On August 9 wrote to Dr. McCracken confirming my agreement to carry out a review of the available literature on epidemiological studies of asbestos and gastro-intestinal malignancies. I requested copies of additional references that I had identified, and these I received towards the end of August.





- 1.4 I have now completed a review of this literature, and have in addition discussed the present stage of the re-analysis of the data from the studies of mortality in the Chrysotile Asbestos Mines and Mills of Quebec, carried out by a team lead by Dr. J.C. McDonald of the McGill University, Department of Epidemiology and Health, with Dr. G. Gibbs of that department, and have also met in person with Dr. I. Selikoff, Director of the Environmental Sciences Laboratory of the Mount Sinai School of Medicine, a visit which was conducted on September 10.
- 1.5 My report consists of three further sections; a review of the available literature, a discussion of this literature and the conclusions I have drawn from my review. I must emphasize that the conclusions I have reached are mine alone, and have not been discussed with any other scientist and in particular, cannot be construed as reflecting the position of either the National Cancer Institute of Canada or the University of Toronto, or any other organization with which I am associated. Indeed, to my knowledge the National Cancer Institute of Canada has never been officially requested to consider this issue, and has never taken up a position on it.
- 1.6 I have taken pains to attempt to ensure, through all possible mechanisms open to me, that relevant literature has been considered. However, I have not carried out a computer assisted search of the literature, and it is possible that some relevant articles have been overlooked. If this proves to be the case, and they are drawn to my attention, I would be pleased to review them and submit a supplementary report or reports, placing them in the context of the literature that I have reviewed and my conclusions from it, as described in this present report. A full list of the literature sources considered is given as an appendix to the report.
- 1.7 The literature on asbestos disease is voluminous, and it would be beyond the abilities of any one scientist to review this in totality within a reasonable period of time. I have therefore, restricted my attention to the literature referring to asbestos and gastro-intestinal malignancies, and ignored reports relating to asbestos and other cancers, including lung cancer, unless it was clearly contributory. I have also ignored anecdotal, or case reports, unless they helped to clarify a point of relevance to the studies under review. Essentially, therefore, I have restricted my attention to population-based epidemiological or statistical studies that have produced data relevant to a consideration of the association between asbestos fibre dust exposure and the subsequent development of gastro-intestinal malignancies.



## 2.0 REVIEW OF THE LITERATURE

- 2.1 Of the references consulted, several were non-contributory in the sense discussed above. Thus, I shall not discuss those numbered 1 to 10 in the appendix.
- 2.2 The second group of references (11-19) consists of those studies in which, although gastro-intestinal cancer was discussed, it was not further considered in relation to site. This, unfortunately comprises much of the literature available on the subject. Although in totality, this might seem to comprise a substantial amount of experience, in practice, this often consists of reports from the same group essentially describing the same series of individuals. Even if it were possible to obtain from the authors a breakdown by site and to attempt a consolidated analysis, it seems unlikely that this would contribute any more to a refinement of the association than those larger studies in which the numbers permitted a breakdown by site within the gastro-intestinal tract.
- 2.3 These literature sources will therefore not be discussed in detail, except to point out that in general their findings are consistent with the more detailed studies, but also to emphasize that without specification by diagnosis, it seems very likely that a substantial proportion of the cases reported may be due to peritoneal mesotheliomas, a problem of diagnosis eloquently discussed by Newhouse and Wagner (19). Indeed, it seems very likely that this difficulty accounted for the earlier suspicion raised by the report of Keal (16) that asbestos was associated with ovarian cancer, as peritoneal mesotheliomas are probably difficult to distinguish from relatively undifferentiated tumours. This indeed seems to have been the opinion of subsequent reviewers (39).
- 2.4 It may be noted that although the studies of Enterline and his colleagues (11-15) seem to ignore this issue, that of Mancuso and El-Attar (17), although they did not separate off different digestive organ sites, for example stomach, colon or rectum, did at least provide data that enables one to determine the numbers of deaths due to malignant neoplasms of the digestive organs if those of the peritoneum were excluded. This study is important because a number of the methodological problems of other studies were understood by these workers, and one of them particularly was tackled by using an internal control, that is, those workers in the same plants with minimal exposure, to develop their expectation for occurrence of disease in those workers with more severe exposure. Although this may not completely remove possible confounding by socio-economic status and other factors, and although it is not clear that they used an ascertainment of confirmation of death that would ensure that all peritoneal





mesotheliomas had been excluded from the other group, it is relevant that their observed expected ratio calculated with the removal of those who died of malignant neoplasm of the peritoneum and/or of mesothelioma is fully consistent with the data from more completely analysed studies, particularly those of Selikoff and others (31-37).

- 2.5 Of the studies in which details of the site of diagnosis of gastro-intestinal malignancies by major organ are given, five provide relatively scant information, either because the cohort under observation was small and/or the data was not sufficiently analysed.
- 2.6 Elmes and Simpson (20) identified 170 men who made up the total population of insulation workers in Belfast in 1940. These men were traced up to 1966, five were untraced and the mortality experience of the remainder was compared with that of other men in Northern Ireland during the same time period. Seventeen deaths due to non-respiratory malignant disease were observed, compared to 5.16 expected using rates from Northern Ireland males. Of the 17, 15 were classified as gastro-intestinal, though 3 of these were mesothelioma and another 1 classified as gastric and 1 classified as rectal carcinoma could have been mesotheliomas. If these are excluded together with the one observed carcinoma of the pancreas and one lymphosarcoma of the small intestine, there still remain three deaths due to carcinoma of the stomach, four to carcinoma of the colon and one from rectal cancer, to compare with an expectation of the 5.16 from all non-respiratory malignant disease considered together. This is obviously consistent with at least a twofold excess of gastro-intestinal cancer excluding mesotheliomas, though further refinement is not possible because of the small numbers. It may be noted that the authors attempted to adjust the excess mortality by factors correcting for social class, domicile and smoking. The adjustment factor for all cancers is 1.22, which if applied to the basic prediction for all cancers in this cohort of 6.84, produces an adjusted prediction of 8.35. As a considerable amount of this adjustment is for factors more relevant to respiratory cancer, it still seems unlikely, if adjustment had been possible for the small numbers of observed compared to expected gastro-intestinal cancers, that the excess would have been eliminated.
- 2.7 Kleinfeld, Messite and Kooyman (21) studied 152 asbestos workers who had 15 or more years of asbestos exposure by 1945, or achieved 15 years of exposure to asbestos dust between 1945 and 1965. The expected mortality was calculated by a proportional mortality analysis using as base year the mortality from U.S. white males in 1948 as this was the median year of death among the 46 deaths observed. At all ages 15.2% of deaths were observed as due to gastro-intestinal and peritoneal malignancies, compared to 3.9% expected. This comprises 7 observed deaths of which 2 were cancers of the stomach, 1 of the colon, 1 a lymphosarcoma of the small intestine, and 3 peritoneal tumours.





- 2.8 This particular report is almost impossible to interpret. Apart from the potential bias of proportional mortality analyses of this type (22), there is an additional bias in that it is not certain that the mortality experience in the cohort was correctly ascertained for comparing with the expectation to avoid the bias noted recently in studies of cohorts exposed to vinyl chloride (23-25). Thus, although the observed proportion is likely to be greater than the expectation if the mesotheliomas and other diagnoses could be excluded, and thus this report would again be compatible with others, no particular reliance could be placed on such a finding.
- 2.9 Knox and others (26) described the mortality experience of 794 men and 220 women, who had been exposed to asbestos fibre dust for a period of 10 years or more in an asbestos textile factory in England. The men were considered in four groups, the majority had exposure to asbestos fibre for 10 to 19 years and no exposure prior to 1933, the remainder had had an exposure for 20 years or more, and were sub-divided according to whether their exposure prior to 1933 was 10 years or more, less than 10 years or none. 1933 was the year in which ventilation systems were introduced, and thus presumably the 57 workers exposed for 10 years or more prior to 1933 had had the greatest dust exposure. They only, however, comprise 918 person-years at risk of death. These were designated group 1 in their report. Expected numbers of deaths were computed from national figures, though dividing up the groups according to intensity of exposure enable an internal comparison to be made. Data of observed numbers compared to the expectation was supplied for all neoplasms with cancer of the lung or pleura separately identified. Excluding the latter group, for the total cohort 14 deaths were observed compared to an expectation of 18.39. In those men exposed for 20 years or more, who had worked at least for some period prior to 1933, the observed numbers were 8 compared to an expectation of 4.99. Details were provided of the cause of death. Of these, 5 occurred in Group 1, one was a cancer of the stomach, 2 colon cancers, 1 gallbladder, and 1 prostate. This total of 5 compared to an expectation of 2.61. It is obvious the expectation of this subcohort, and possibly the period of observation of the other cohorts was insufficient to confirm or refute the findings of others, particularly that of Selikoff and his group (31-37) though it may be noted that the findings in this study were confirmatory for lung cancer.
- 2.10 Mancuso and Coulter (27) reported on a cohort from an asbestos company identified through the use of Bureau of Old Age and Survivors Insurance Data. The cohort comprised individuals employed at any time during the years 1938 to 39. These were



followed through the BOASI source to mid 1960. Person-years at risk were computed and compared for selected underlying causes of death on the basis of death rates from the state of Ohio from 1950 to 1960. Ohio data was used as corresponding data, for the state in which the plant was located was not available and the period 1950 to 1960 was used instead of 1940 to 1960 because of the unavailability of earlier data. It was believed that Ohio death rates reflected the expectation from the corresponding state though it was noted that any excess in observed deaths over expected deaths due to specific causes of mortality would be underestimated for causes of death with rising mortality trends and somewhat over-estimated for causes with declining mortality trends because of the use of mortality rates from the second part of the observed 20 year period. This would have been important if an excess of deaths from carcinoma of the stomach had been claimed. In practice this was not so, although a detailed tabulation showed that the observed numbers (with the expected in brackets) for digestive organ and peritoneum were 18 (8.07), while by major organ the breakdown was esophagus 1 (0.53), stomach 2 (1.87), large intestine except rectum 3 (2.29), and rectum 2 (1.15). This report, however, only permits a maximum 20 year follow-up and no data was supplied to permit analysis by duration of employment or intensity of exposure. Furthermore, the study was restricted to ages 25 to 64 and the authors themselves felt that the order of excess noted for lung cancer 19 (5.61) and peritoneal cancer 3 (0.08) were conservative estimates. Nevertheless, this must be regarded, as far as it goes, as a largely negative study.

- 2.11 Meurman, Kiviluoto, and Hakama (28) studied 1092 asbestos workers first employed at 2 anthophyllite asbestos mines between 1936 and 1967. Follow-up was to June 1969, 95% of the workers being traced, 248 of whom had died. A unique feature of this study was that not only were the deaths observed compared to an expectation for national mortality data (the year 1958 was used as the mid-year of deaths in the cohort), they also selected a matched control cohort of individuals residing in a town 60 kilometers northwest of the mining community during the period 1936 to 1967, using a local population registry. Seven digestive organ cancers were observed in the asbestos workers, 9 in the control cohort compared to an expectation of 14.9 from national rates. There was no excess in any age group, for example, at ages 65 to 74 the corresponding numbers were 4, 4, and 4.2. In a subgroup of employees, exposed for 10 years or more, 2 deaths from digestive system cancer were observed compared to 2.1 expected. It may be noted that in a comparable analysis for lung cancer there were 8 cases observed compared to 2.4 expected. Thus, it would seem that this study has failed to show any evidence of an excess of digestive organ cancer even though the cohort superficially seems reasonable in size. However, this may be a spurious assessment because those who most





recently entered the cohort can have only had a 2 year period of observation and it seems likely that the numbers of workers who had a 20 year period of follow-up is likely to have been very small. As reviewed below, it is only this group which showed the excess in the studies of Selikoff and his colleagues (37). Furthermore, it is possible that using a single year for deriving expected rates may have over-estimated the expectation, especially for stomach cancer, which has been declining in mortality during the time period considered. Thus, although this must be regarded as a negative study, it is still possible that further follow-up of the cohort might reveal findings similar to those of other workers.

- 2.12 There remain studies by 2 group of workers which seem to be more contributory to the subject under consideration.
- 2.13 McDonald and his colleagues have produced 2 reports on mortality experience of workers in the Chrysotile Asbestos Mines and Mills of Quebec (29, 30). The cohort identified for the study comprised 11,788 persons born between 1891 and 1920, who had been employed for one calendar month or more. These were then traced by various mechanisms in order to establish live or dead status as of November 1, 1966. Information was obtained concerning 10,421 (88.4%) of the cohort. Two thousand four hundred and fifty-seven (23.6% of those traced) were reported dead. For the majority of these copies of death certificates were obtained and for those suspected as dying of lung cancer further information was sought. No particular tracing seems to have been employed for cases registered as dying from gastro-intestinal cancer. Of the total cohort, 1,203 had worked 30 years or more, while 3,738 had worked for less than one year and an additional 1,080 had worked for more than one year, but had had low dust exposure. A "dust index" for exposure was determined and the analysis was performed by calculating what was described as equivalent average death rates per thousand men for different categories of malignant neoplasms according to the dust index. These rates were age standardized. Table 4 of the 1971 report (29) indicates an increasing trend of mortality for intestinal or rectal cancer by dust index, the ratio between the highest and the lowest categories being 3.5, but an inconstant trend for esophagus and stomach cancer considered together mainly because the lowest category of dust index had almost as high a mortality rate as the highest, the ratio being 1.3 even though those with a dust index towards the middle of the scale had a substantially lower mortality than either of the two extremes of the distribution. A comparison with Quebec mortality rates was carried out for all causes of death and for lung cancer, but if performed for gastro-intestinal cancer, was not reported. For all causes of mortality,





the expected numbers of deaths were 1,824 in men compared to 1,674 observed, while for lung cancer, the numbers were 91 and 94 respectively. In the discussion of results in the 1971 paper, the authors indicate that they feel that an excess of lung cancer in most heavily exposed categories of workers might be of the order 3 to 5 fold, possibly substantially less than that reported by Selikoff and his colleagues. They did not attempt to further interpret the findings for gastro-intestinal cancer.

2.14 In a subsequent paper (30), no additional data was presented, however, they did state "the primary method of analysis used in our report on deaths up to 1966 had 2 main weaknesses. The first was that length of the exposure might well have been related to length of survival and thus might tend to obscure differences in mortality between exposure groups -- The second lay in the fact that deaths accumulated over many years were used in a single calculation of mortality." Thus the authors have admitted that their analysis had many of the potential pitfalls of such analyses (23-25). Nevertheless, it is difficult to see how a significant trend for colo-rectal cancer within the different exposure categories could have been introduced by the errors admitted. Rather the comparison with Quebec mortality will have underestimated the magnitude of the increased mortality due to asbestos exposure. Clarification of the present position with Dr. Gibbs (Personal Communication) elicited the information that deaths up to the end of 1974 had been ascertained and are currently in the process of analysis, however, it is not anticipated that any results will be available until the end of this year at the earliest. It may be noted that in their 1974 report, the authors indicate that the analysis they propose will be based on man-years of exposure to minimize those errors noted. It is unfortunate the data is not available from this cohort in the detail and format that would be required for proper interpretation as this is probably the largest experience likely to become available in the foreseeable future.

2.15 The second group of workers that have reported extensively, but generally speaking, repetitively on the same cohorts is that of Selikoff and his colleagues (31-37).

2.16 The publications of these workers have been frequent and, in addition, particularly in recent years, earlier papers have been updated in a number of ways including presentations at meetings, the full details of the presentation having not always been subsequently published. Furthermore, presentations at meetings often do not permit adequate description of details of ascertainment of studied individuals and their subsequent follow-up and the analysis of their experience. This has caused some difficulty in interpretation and at times has lead to the suspicion that the data may not be as firm as the published claims or the claims made in presentations may have indicated. The doubts engendered by this are exemplified in the report of Dr. Ritchie. It was for this reason that I have been at pains to attempt to identify all relevant published materials by these authors and also made a personal visit to clarify the questions raised in my mind by a review with Dr. Selikoff of this material.



- 2.17 Three differing, but in one respect overlapping cohorts have been identified and studied by these investigators. The first is a cohort of 632 asbestos insulation workers, who were members of the New York and New Jersey locals of the asbestos workers union, that is locals 12 and 32 of the International Association of Heat and Frost Insulators and Asbestos Workers. These men had all entered employment prior to December 31, 1942 and were alive on that date and in the initial publications were followed through to December 31, 1962 (31, 32). Coincidentally, as this cohort of 632 workers was identified another 890 workers, who entered the relevant union locals between January 1, 1943 and December 31, 1962 were also identified, but these have never been the subject of an independent report except to the extent they contribute to the reports on the third cohort to be described. Subsequently, a subsegment of the cohort of 632 workers comprising 370 who were alive and examined and followed from January 1, 1963 were described as being of particular interest with regard to the interaction between smoking and asbestos exposure and lung cancer (33, 34). This particular subsegment, however, though having their experience in relation to gastrointestinal cancer described, is not of particular value with regards to the question of prime interest in this report and they will not be referred to further.
- 2.18 The second cohort comprised 933 Amosite factory workers, who started work in a single factory some time between June, 1941, when the factory began production and December, 1945. The plant continued production until November, 1954, when it closed its doors and in the first publication, the population was followed through to June 30, 1971 (35).
- 2.19 The third cohort comprised the entire membership of the International Association of Heat and Frost Insulators and Asbestos workers of the United States and Canada on January 1, 1967, comprising 17,800 workers who also included those members of cohort one that were still alive on the date of ascertainment. This cohort has never been described in an independent publication, but was referred to in 2 reports which served also to update the experience of the other 2 cohorts. The first of these (36) was presented at the meeting of the working group to assess the biological effects of asbestos sponsored by the International Agency for Research on Cancer in Lyon, France, October 4, 1972. This paper was published in the volume reporting the discussions of the working group. This updated the experience of all 3 cohorts to December 31, 1971. The second update has been published in the proceedings of a meeting to consider persons at high risk of cancer and updated the experience of all three cohorts to December 31, 1973 (37). Subsequently, the experience of the cohorts has been updated to December 31, 1974 and copies of the tables detailing this were supplied to Dr. Ritchie and appended to his report and were discussed by me





with Dr. Selikoff during my visit on September 10, 1976. In discussing the experience of these cohorts, unless otherwise indicated, it will be the results of this update to December 31, 1974 that will be utilized.

- 2.20 The ascertainment and follow-up of all three cohorts essentially followed an identical pattern and may be summarized as the identification of a total group of occupationally exposed workers within defined limits, who are established as being alive on a date subsequent or immediately following the last date of the period of time used to establish the cohort. It is important to note that this procedure removes nearly all the potential biases that have been described by others (23-25). All were subsequently followed by periodically confirming that the men were alive and for those who were dead eliciting copies of death certificates, tracing hospital records wherever possible and collecting histological material for the large majority where it seemed likely that a cause of death of interest had in fact occurred. Considerable care was taken in this review of the cause of death to confirm that certified. Indeed, where it was not possible to obtain detailed information, preferably histological, though it seemed likely that an individual had died of malignant disease, then the case was categorized as dying from other cancer and included in the tabulations under the heading "All other cancer". This certainly explains the reason why there is an excess of observed over expected deaths under this category, but also this may serve to underestimate the difference between observed and expected in other categories to the extent that individuals who in fact died of, for example, stomach or colorectal cancer were incorrectly tabulated under all other cancer. Thus the reported observed/expected ratios are conservative. The calculation of expected deaths was based on age specific mortality data derived from the United States National Centre for Health Statistics. To the extent that these reflect a different expectation than the cohort then the results of the analysis will be in error. For example, it might be anticipated that a group of New York, New Jersey workers might not necessarily have the mortality experience identical to that of national rates while it is not clear the extent to which U.S. national rates are similar to Canadian rates for the expectation of deaths for that relatively small portion of the third cohort which was established and living in Canada. The effect of an error from this source, however, is likely to be very small. A possibly more basic criticism is the fact that all comparisons are based on expected rates derived from national data and no control from another source was established for any of these cohorts. For some of the data, internal controls are, however, available, especially when analysis is performed according to





duration of employment. Duration of employment has in fact been used by these investigators as the closest approach to determining a dose response relationship. This is because most of the workers studied performed a number of different jobs, the nature of which could not be clearly specified in terms of intensity of dust exposure.

- 2.21 The updated results can be summarized as follows. In cohort one, the 632 workers, 20 cases of carcinoma of the esophagus or stomach were observed with an expectation of 6.5, and for carcinoma of the colon 23 (7.6).
- 2.22 In cohort two, the 933 Amosite workers, gastro-intestinal cancer is not broken down in the 1974 update. Twenty-four cases were observed, 12.05 expected. Substantially more detail is available in Table 4 of (37), where in total 10 cases of carcinoma of the stomach were observed with 4.89 expected and 16 of carcinoma of the colon and rectum, 7.65 expected. It may be noted that in this table, the ratio of observed to expected tabulated for cases before 1953 for carcinoma of the stomach is printed as 7.78, but this is an error. It should be 1.78. It may also be noted that the observed to expected ratio increases the longer the period since the establishment of the cohort. Table 8 of the same publication considers occurrences according to duration of employment for carcinoma of the stomach, a clear trend is observed of increasing ratios of observed to expected with increasing duration of employment, though these are based on small numbers. For carcinoma of the colon and rectum, all the ratios show an excess of observed compared to expected, though in this instance a trend is not seen.
- 2.23 In cohort 3 (the 17,800 workers) the numbers permit a breakdown according to observation periods for less than 20 years from onset of work and more than 20 years from onset of work. In the former category with 12,683 men and 77,461 man-years of observation, the numbers of observed (with the expected in brackets) for the relevant categories are esophagus cancer 1 (0.58), stomach cancer 1 (1.37), colorectal cancer 4 (3.40). Thus no increased risk was demonstrated. For those 10,967 men who had been observed for 20 years or more from onset of work, there were 57,825 man-years of observation and the numbers in the relevant categories were esophagus cancer 13 (4.77), stomach cancer 17 (9.86), colorectal cancer 43 (25.23). The differences for esophagus and colorectal cancer were highly significant ( $P=.001$ ), while that for stomach cancer attained conventional significance levels ( $P=0.05$ ).



### 3.0 Discussion

- 3.1 There is no doubt that an association has been demonstrated between exposure to asbestos and the subsequent development of cancers of the gastro-intestinal tract, particularly that of the esophagus, stomach, colon and rectum. The important question is the extent to which this association can be interpreted as being causal.
- 3.2 A number of authors have described the criteria which should be applied before deciding that the most likely interpretation of an association is causation. Perhaps one of the clearest of these expositions is that of Bradford Hill (38) and I shall discuss this issue according to the headings he uses.
- 3.2.1. Strength: There can be little doubt that the demonstrated association between gastro-intestinal cancer and asbestos exposure is weak. The risk ratios quoted, i.e. the ratio between the observed and the expected deaths, rarely seem to exceed 3 and in many instances are around 2. The worry over weak associations is the possibility that they may be explicable on the basis of other factors whose relevance was not considered either in the study design or in the subsequent analysis. The most obvious example is socio-economic status and, indeed, as most of the comparisons that have been conducted are between the observed mortality in an identified cohort and an expectation based on national mortality rates, this might be expected to confound the issue. Nevertheless, it may not be true to anticipate that this would necessarily operate in the direction of causing a spurious association to appear. Thus, asbestos insulation workers are not necessarily unskilled, falling more under the category of skilled workers such as might be categorized under the British Registrar General's category of 3 rather than 4 or 5. This might indicate an expectation of mortality very similar to the national average. In addition, although it might be expected that in a low socio-economic group, stomach cancer might be higher than the national expectation, the reverse would be anticipated for colon and rectal cancer, yet both appear to be increased in most studies to almost the same extent. It seems difficult, therefore, to believe that socio-economic status should be a cause of a spurious association when different directions of effect in these 2 cancer sites would be anticipated. Furthermore, in those studies where either internal controls have been utilized or control has been attempted for socio-economic status, the result has not seemed to explain the differences observed.





- 3.2.1.1 In many respects use of national mortality data to derive expectation may underestimate the magnitude of the difference and thus the strength of the association. It has been frequently observed that occupational groups have lower all-causes mortality (other than those possibly associated with the occupation) than would have been expected from the general population. This has been almost universally the experience of cohorts studied for disease experience subsequent to asbestos exposure when causes other than those suspected or established as due to this exposure, were considered. Nevertheless, it has not so far been established how a correction for this "healthy worker effect" could be applied.
- 3.2.1.2 In his discussion of this criterion, Bradford Hill points out "We must not be too ready to dismiss a cause and effect hypothesis merely on the grounds that the observed association appears to be slight." Although this seems to have been the grounds an advisory committee on asbestos cancers convened by the International Agency for Research on Cancer (39) failed to conclude that a causal relation existed, they did not discuss all the criteria that should be applied and indeed met at a time when some of the more recent evidence was not yet available.
- 3.2.2. Consistency: As already discussed, a number of the studies reported, which failed to show the association, seemed either to be too small or possibly to be observing a cohort for too short a period since exposure to expect that an association might be demonstrated. For those studies which do not seem to have such limitations, even those which do not specify gastrointestinal cancer by organ of origin, the criterion of consistency seems to be met. Although most of the studies were analysed using a similar approach, a number of different cohorts are involved, some exposed to different types of asbestos and in different countries and for this reason the consistency criterion is important.
- 3.2.3. Specificity: This characteristic essentially demanding an almost one to one relationship between a suspected causal agent and the ensuing disease is obviously not met, thus asbestos is accepted as an etiologically relevant agent for one type of pulmonary fibrosis and in malignancies for lung cancer and for the very different pleural and peritoneal mesotheliomas. With regard to gastro-intestinal cancer, the association exists for esophageal, stomach, and colorectal cancer, though with regard to the latter, it is not particularly clear as to whether or not the association has been demonstrated for rectal as distinct from colonic carcinoma. Few studies enable the data to be distinguished on this point, though it seems certain that the association is present for colonic carcinoma.
- 3.2.3.1 However, as Bradford Hill points out, "one to one relationships are not frequent. Indeed, multicausation is generally more likely for a disease than single causation" so that the lack of specificity does not necessarily rule against causation.





- 3.2.4. Temporality: This fourth criterion is designed to distinguish between those associations which arise because of or after a disease has occurred or which clearly preceeded the disease in a reasonable causal chain of events. As all the studies reviewed have been essentially the same type, namely cohort or prospective, even if some of the cohorts were established in retrospect, the temporality of the association has been clearly demonstrated.
- 3.2.5. Biological Gradient: This criterion is most clearly expressed in terms of a dose/response relationship. As indicated in the review, there are few studies which enable this to be determined. The one that most clearly tackles this issue is that of McDonald and his colleagues (29, 30), though as already indicated, even the authors of these reports accept that their analysis may have been faulty. However, the errors in the analysis are of a type which it seems likely will have underestimated the strength of the association or resulted in failing to demonstrate a dose/response relationship. It is difficult to visualize that the errors in the analysis would have created the dose/response relationship for colorectal cancer and this must be regarded as a strong point in favour of regarding this particular association as causal. Furthermore, other approaches to attempting to obtain dose/response relationships such as duration of exposure or period since initiation of employment contribute to the suspicion that a dose/response relationship does in fact exist.
- 3.2.5.1 Historically, the suspicion that asbestos exposure was causally related to lung cancer was delayed in its confirmation because insufficient time was allowed both to ensure that those individuals in the studied cohorts had acquired sufficient exposure and that they had been observed for long enough to exceed the "latent period" between initiation of exposure and development of disease. The studies of several workers, particularly those of Selikoff and his colleagues, appear to confirm the presence of a latent period which is probably 20 years or more (37). For this reason, it seems possible that the largest cohort under observation by Selikoff (cohort 3, the 17,800 workers) has yet to experience it's greatest mortality from gastro-intestinal cancer. Indeed, in many respects, this is a far younger cohort than the other two studied by these investigators and McDonald and his colleagues.
- 3.2.5.2 Even so, the various observed/expected ratios reported by Selikoff do not indicate any consistent trend to increasing ratios with further follow-up of the early cohorts established by this group. Nevertheless, this may not necessarily be expected in studies in which essentially the same small group of workers are repetitively reanalysed.



3.2.5.3 It may, however, be noted that in more recently established cohorts, the expectation for death from gastro-intestinal malignancies, or indeed any other asbestos related condition, may fall if there is a threshold effect for induction of these diseases and if measures taken almost universally in asbestos related industries to reduce exposure to asbestos dust have reduced the exposure of the majority of workers below the threshold. This indicates that it is highly desirable to study not only existing cohorts already under observation, but to attempt to establish more recently exposed cohorts so that if the risk is reduced to the extent that no further excess is demonstrated after suitably prolonged periods of observation, such absence of risk can be documented. Indeed, one of the important aspects yet to be established in relation to asbestos and cancer that has been clearly demonstrated in relation to smoking in the absence of asbestos exposure and lung cancer is the fact that the disease no longer occurs in individuals in whom exposure has ceased. Nevertheless, as asbestos appears to persist in the tissues and possibly to act in this regard either as solely an initiating agent or as both an initiating and a promoting agent, whereas smoking clearly acts as a promoting agent alone, then expectation of a reduction of risk in individuals once exposed may be unrealistic in the biological sense.

3.2.6. Plausibility: In Bradford Hill's words "It will be helpful if the causation we suspect is biologically plausible, but this is a feature I am convinced we cannot demand. What is biologically plausible depends on the biological knowledge of the day."

3.2.6.1 Data on this criterion cannot be obtained through epidemiological studies, however, the data that is available and was reviewed by Dr. Ritchie would support the biological plausibility of the suspected association. Even though asbestos is generally inhaled, it may be anticipated that those who are exposed to inhalation would tend to trap many asbestos particles in the sputum and then these would then expose the gastro-intestinal tract when the sputum was swallowed. Indeed, unless all peritoneal mesotheliomas develop through penetration of fibres through the diaphragm, it is difficult to see how they could arise, except through penetration through the gastro-intestinal tract. Thus it would seem to be plausible to anticipate, even in the absence of demonstrated fibres in tumours, that swallowed asbestos fibres are carcinogenic, by whatever biological mechanism in fact operates, in all parts of the gastro-intestinal tract.

3.2.7. Coherence: In Bradford Hill's words, "On the other hand, the cause and effect interpretation of our data should not seriously conflict with the generally known facts of the natural history and biology of the disease." Coherence in this sense is difficult





to establish in the absence of information on the etiology of most cancers of the gastro-intestinal tract other than suspicions of the effect of highly spiced or irritant foods in producing gastric cancer or high fat or animal protein diet in producing colorectal cancer. Whether or not asbestos is acting in association with such factors cannot be determined on present knowledge. Thus, although it is obvious that asbestos exposure is not necessary for the development of gastro-intestinal cancer, we cannot determine whether alone it is sufficient or whether it acts in collaboration with other etiological agents. By analogy with lung cancer, it seems possible that asbestos may require to act in an association with another agent (smoking for lung cancer), though knowledge is just not available on this point.

- 3.2.8 Experiment: This criterion is used by Bradford Hill not in the sense that animal experimental data may help, he presumably relates this to biological knowledge; (in passing, it may be noted that animal experimental data on causation of gastro-intestinal cancers through asbestos exposure is still controversial), but in regard to the possible effect of an experiment in the human situation, thus the strongest evidence might come if it were possible to remove exposure to an etiologic agent and the disease would cease. The difficulties over this in relation to asbestos have already been alluded to, at least in terms of already exposed individuals. Furthermore, it seems unlikely in view of the ubiquity of exposure to asbestos that any occupational group that uses it could in fact be completely prevented from exposure of their lungs or gastro-intestinal tract. Thus, apart from the difficulty of long term observation that would be required, expectation of establishment of causation on this criterion may be somewhat academic.
- 3.2.9. Analogy: The final criterion used by Bradford Hill is the reasonableness of ascribing causation, if in fact a similar mechanism has been shown to operate for another condition. In practice, asbestosis is so unique that it seems unlikely that one would be able to demonstrate a similar chain of events in relation to gastro-intestinal cancer. The nearest one could come to analogy is that of the occurrence of lung cancer and pleural and peritoneal mesotheliomas. However, this does not seem to be the sense in which Bradford Hill uses this criterion.
- 3.3 Two further points require comment. Firstly, in his original letter to me, Dr. McCracken indicated that one of the difficulties of the literature was the absence of data relating to exact anatomical sites of cancers. Although it would be nice to be in a position to identify cancers of particular sites within the





stomach or other parts of the gastro-intestinal tract or even more so a particular histological variety that would facilitate identification of clearly asbestos related cancers, data is just not available on this point and it is unrealistic to expect that it will become available from existing methods of epidemiological study. I am informed by Dr. Selikoff that so far it has not been possible using anatomical or histological criteria to clearly identify those cancers of the gastro-intestinal tract that are due to exposure to asbestos. Nevertheless, in view of the nature of the agent, it may be unrealistic to separate off gastro-intestinal cancers even by organ. If one is dealing with a statistically rare event, and if the nature of the agent is such as to cause fairly uniform exposure to the gastro-intestinal tract, then maybe all we should expect is a general increase in the incidence of those cancers which normally occur. That the esophagus is not preferentially at risk compared to the stomach and the stomach compared to the colon seems to be established on the data that is available. Furthermore, the marked excess of peritoneal mesotheliomas in all series seems to suggest that in many, if not most instances, asbestos exerts its' neoplastic potential when it comes to rest in the peritoneum and that possibly the rule is that either it is excreted or that it passes through the epithelium lining the gastro-intestinal tract and only rarely lodges for a sufficient period of time to induce neoplasia. If this is so, then what may be anticipated is an increase to a certain degree in the incidence of cancers that normally occur and in general all studies are supportive of this.

- 3.4 Secondly, it should be noted that the type of methodology normally utilized in occupational studies is such as to facilitate identification of strong associations (such as has lead to the demonstration of a causal association of asbestos and lung cancer, especially in the presence of smoking), but which does not facilitate the refinement of the nature of the relationship, especially if the association is relatively weak, as in the present case. Although further data can be anticipated in the years to come from continued observation from the cohorts studied by Dr. Selikoff and his colleagues and the reanalysis of the data of McDonald and his colleagues, the type of information that will be obtained is already known. Even the analysis of the large occupational cohort, identified through the 10% sample of the Canadian labour force from 1965 to 1968 and a 5% sample from 1969 to 1971, to be linked with national vital statistics data and national cancer registration data in a study being conducted by the Epidemiology unit of the National Cancer Institute of Canada, may not include sufficient asbestos workers who have been exposed long enough to further clarify the issue. Therefore, it seems likely that the data currently available is as good as may become available in



the future unless extremely detailed studies of large cohorts carefully categorized by intensity of exposure, preferably with control through other occupational or general population groups, prove possible in the near future. It may be noted that no such studies are being conducted as far as I am aware and that even the study in Finland (28), which has some of the features of an ideal design, and from which further confirmatory evidence may become available after another decade of follow-up, appears to lack detailed information on extent of exposure.

- 3.5 A decision, therefore, has to be taken on the basis of available evidence and on the basis of weighing the data that is available in spite of the acknowledged deficiencies already discussed in this report.

#### 4.0 Conclusion

- 4.1 Exposure to asbestos is associated with the subsequent development of gastro-intestinal malignancies. In the absence of any explanation to the contrary, this exposure must be regarded as causal.
- 4.2 There appears to be a latent period before the effect is demonstrable. Thus on present evidence, only those cases that are identified as developing gastro-intestinal malignancies after a period of 20 years or more from first exposure should be accepted as being due to the exposure.
- 4.3 All anatomical sites of the gastro-intestinal tract appear to be affected.
- 4.4 The degree of increased risk of gastro-intestinal cancer in asbestos workers 20 years or more following first exposure approximates to 3 fold.
- 4.5 There is no evidence that any other factor either contributes to or subtracts from the causal relationship between asbestos exposure and cancer of the gastro-intestinal tract.

September 23, 1976

Signed



A.B. Miller, M.B., FRCP (C)



APPENDIX

List of literature sources considered.

A. Not Contributory

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ASBESTOS AND CARCINOMA OF THE LARYNX

PRELIMINARY REPORT, APRIL 1978

BY DR. A. B. MILLER, M.B., FRCP(C)

Introduction

In October and November 1976 consultations were held between myself, Dr. W. J. McCracken of the Workmen's Compensation Board, Dr. A. Ritchie of Toronto General Hospital and Dr. D. Bryce of the same hospital as to whether or not it would be practicable to clarify the possible causal relationship between asbestos and cancer of the larynx by further epidemiological studies. I advised that a case control study based on newly diagnosed cases with a carefully designed questionnaire giving information on occupational and other exposure might well serve to clarify the relationship. Further, a study restricted to southern Ontario (primarily based on cases newly diagnosed and identified through the Princess Margaret Hospital and the Hamilton Cancer Clinic) should provide a sufficient sample of cases to confirm or otherwise the substantially increased risk suggested by a study conducted by Dr. Morgan and Mr. Shettigara, a graduate student working under Dr. Morgan's supervision (see below). However, I also warned that it might take a two-year period of case and control acquisition to adequately answer all questions raised by previous studies of cancer of the larynx and occupational exposure. At the request of the Workmen's Compensation Board the study was designed to obtain information not only on asbestos exposure but on exposure to nickel and after reviewing a document outlining the proposed study the Board agreed in February 1977 to support for one year the expenses incurred in obtaining and entering the data on our computer, namely the salary of a full-time interviewer, the costs of her travel, and the costs of data coding and entry.

After pre-testing the questionnaire; obtaining agreement from the Princess Margaret Hospital, the Hamilton Cancer Clinic and physicians who treat laryngeal cancer that we could identify cases; recruiting and training the interviewer; case acquisition and control identification commenced in May 1977. In order to comply with a request that a preliminary report be made available to the Workmen's Compensation Board by the end of March 1978, although case and control identification is still proceeding only that data complete and available to us by the end of January was included in the analysis which is the basis of the present report. This comprises a total of 62 case control pairs.

Previous Studies

Stell and McGill (Lancet 2, 416-417, 1973) questioned 100 male patients with squamous carcinoma of the larynx and 100 matched controls with various non-malignant diseases. Thirty-one patients and three controls had experienced "an important degree of exposure to asbestos." The difference in exposure history between the groups was highly significant. Subsequently Newhouse and Berry, (Lancet 2, 615, 1973)





reported that among a cohort of over 4,000 workers followed since 1933 there had been two deaths from cancer of the larynx compared to 0.37 expected from the mortality from this cause among the male populations of England and Wales.

Pedersen, Hogetveit and Andersen (International Journal of Cancer 12, 32-41, 1973) reported on cancer of respiratory organs among workers at a nickel refinery in Norway. Among 1,916 men observed for various periods five cancers of the larynx were observed compared to 1.29 expected. The authors commented that these cases permit no firm conclusions but that this may be another manifestation of risk due to occupational exposure. Four of the cases were noted among process workers (roasting and smelting). As far as I am aware this is the only report indicating a potential risk for cancer of the larynx among workers exposed to nickel.

Shettigara and Morgan (Archives Environmental Health 30, 517-519, 1975) reported on 43 case control pairs, the cases being identified through the Toronto General Hospital and the controls being neighbourhood controls matched for sex and age, of whom ten patients reported exposure to asbestos and no controls. Subsequently, this study was updated (Annals of the New York Academy of Sciences 271, 308-310, 1976) providing information on 54 case controls pairs of whom 13 of the cases were exposed and one of the controls. All cases and controls had been interviewed by Mr. Shettigara and in practice the questionnaire was rather brief and rather specifically directed to asbestos. However, information was obtained on smoking history and it was noted that the risk imposed by asbestos was confined to smokers.

Selikoff and Hammond (In "Persons at High Risk of Cancer" edited J. F. Fraumeni, Academic Press, New York, 1975 pp. 467-483) in a paper largely devoted to consideration of the interaction between asbestos exposure and lung cancer, nevertheless indicated in one table that among their cohort of 17,800 asbestos insulation workers 4 cancers of the larynx had been observed whereas less than 2 had been expected. This information was given for those for whom smoking histories were known. Although this data has never been subsequently up-dated in publication Dr. Selikoff has up-dated the findings relating to laryngeal cancers specifically and has made these tables available initially to myself then to Dr. McCracken. In a table giving the experience of the cohort up to December 31, 1975 nine deaths from cancer of the larynx were observed compared to 4.46 expected. The excess of observed and expected, however, was almost entirely restricted to that group of 6,143 workers for whom a smoking history was unknown. Subsequently Dr. Selikoff up-dated this information in a slightly different form but we understand that the observed cases are now eleven though the excess still appears to be in the cigarette smoking unknown group, the observed to expected ratio being 6/1.44 or 4.2. It was this anomaly within categories which suggested that some other factors might be operating as well as the exposure to asbestos among the smoking unknown group and which advised caution over accepting these results at their face value.

Wynder and his colleagues (Cancer 38, 1591-1601, 1976) evaluated environmental factors in cancer of the larynx in a case control study. Two hundred and fifty-eight men and fifty-six women with cancer of the



larynx were studied and compared to a matched control group hospitalized in the same institution as the case at a ratio 2 to 1 for males and 3 to 1 for females. An excess risk for tobacco consumption and alcohol intake was confirmed and although it was noted that there was an excess of occupational exposure and that some of the construction workers may have been exposed to asbestos, no attempt to quantify such exposure was made nor to develop an appropriate estimate of risk.

Newhouse (Personal communication 1977) has conducted a case control study in London, England. This apparently has revealed no evidence of increased risk from exposure to asbestos, but, in spite of a number of requests, no data have been made available to me.

#### Present Study

The present study is of case control design based on newly diagnosed cases with laryngeal cancer in southern Ontario and the Sudbury region of Ontario with the prime objective of investigating the relative contribution of known and suspected etiological factors for laryngeal carcinoma. A prime focus of the study has been to evaluate possible occupational causes, specifically asbestos exposure, but also possibly other occupational factors such as exposure to nickel and nickel manufacturing processes. The questionnaire is administered in the home of the case and of the matched control obtained by door to door searching in the neighbourhood of the residence of the case. A detailed occupational history is taken and a detailed history of use of tobacco and alcohol. Towards the end of the questionnaire specific questions are asked over exposure to asbestos and in a separate section exposure to nickel and at the end of the questionnaire the respondents are asked as to their knowledge over possible etiological factors for cancer of the larynx.

Sixty-two case control pairs have been included in the analysis but it was found that only two cases and one control mentioned asbestos exposure during the detailed occupational histories. The corresponding numbers for nickel exposure were two cases and no controls. Hence if the analysis were to be restricted to the information obtained from the occupational history section of the questionnaire the study at present would have to be regarded as being non-contributory with regard to exposure to both asbestos and nickel.

On the other hand a much larger number of subjects responded positively to the specific questions on asbestos and nickel exposure and further analyses have, therefore, been based on these responses. Before summarizing the findings from this section of the analysis, however, it may be noted that we were particularly aware of the risk that the answers to these specific questions might be biased because of the greater probability that cases recall such exposures than controls. Restricting the analysis to those cases and controls who denied any knowledge as to the causes of laryngeal cancer did not alter the findings and, therefore, the full results are reported. Furthermore, questioning of the interviewer did not indicate any reason to suspect bias in the efficiency with which information was obtained, while the





duration of the interview was similar for cases and controls.

Of the sixty-two cases fourteen have a history of exposure to asbestos compared to nine of the sixty-two controls. The discordant pairs ratio was 13/8 for a risk ratio of 1.6.

For exposure to nickel, eight of the sixty-two cases gave a history of exposure compared to one of sixty-two controls. The discordant pairs ratio is 8/1 for a risk ratio of 8.0.

On the basis of ever versus never exposed, therefore, the risk ratio for asbestos exposure is non-significant, but significant for exposure to nickel ( $p=0.02$ ). However, three of the cases gave a history of exposure to both asbestos and nickel and if these are eliminated from consideration the risk ratio for nickel exposure becomes five and is non-significant.

These results have been presented without any attempt to rank the intensity of the exposures. However, as asbestos is fairly ubiquitous this would not seem to be a satisfactory basis for analysis of its possible effects. Perusal of the histories given by the respondents (without consideration of their case or control status) indicated that a number of those exposures recorded to asbestos were non-occupational. None of the exposures to nickel fell within this category. However, in addition it seemed that some of the occupational exposures might be regarded as being of little importance. An attempt has, therefore, been made to grade the intensity of exposure as reported on the questionnaire into three categories: 1, non-occupational and minimal, 2, occupational and minimal, 3, occupational and non-minimal. Three observers familiar with the study within the NCIC-Epidemiology Unit were asked to categorize all cases and controls in this way without knowledge of their case control status. The resultant categorization was remarkably uniform and Table 1 sets out, for both asbestos and nickel, the relevant findings. It is apparent that the excess of cases with asbestos exposure is restricted to those in category 3. The risk ratio for those in this category as compared to those never exposed is 9/3 i.e., 3.0 ( $p=0.07$ ). The risk ratio for nickel exposure is barely changed becoming 7/1 ( $p=0.04$ ). Hence, exposure to asbestos when considered on the basis of important occupational exposure is now on the borderline of statistical significance. Of the three cases who had joint exposure to both asbestos and nickel, all were regarded as having category three exposure to asbestos, 2 as category three exposure to nickel and 1 as category two. As before if these are eliminated from consideration the findings for both asbestos and nickel become non-significant.

In an attempt to clarify the role of asbestos versus nickel in the three cases with dual exposure, the data available in all questionnaires was listed and reviewed independently by an occupational hygienist and by an occupational medical specialist (M.D.) in the occupational Health Unit of the University of Toronto. They attempted to code all the exposures in the same way as had been coded within the NCIC-Epidemiology Unit. This, unfortunately, did not clarify the issue. One of the patients who recorded exposure to nickel mixed with steel from 1940 to





1976 and worked as a furnace man with asbestos coats on furnaces from 1940 to 1945 was classified 3 on both counts by the occupational hygienist though 1 and 3 respectively by the medical specialist. A second, who hauled nickel oxide from 1963 to 1970 and over the same period acted as a pipe fitter with exposure to asbestos insulation over pipes in homes was coded as 3 for the nickel exposure and 2 for the asbestos exposure by the hygienist though 1 and 2 respectively by the medical specialist. The third who acted as a supervisor with exposure to stainless steel sheets from 1929 to 1975 who had regarded this as exposure to nickel and also was exposed during the same period to asbestos sheets used as sound deadeners in elevators was classified on both exposures 2-3 by the hygienist though 1 and 3 respectively by the medical specialist.

A number of the exposures were placed in mixed categories by the hygienist and Table 2 indicates the results of a case control analysis using his ratings. Table 3 gives the corresponding analysis for the ratings by the medical specialist. It becomes obvious that he tends to rate nickel exposure low, but asbestos high. Nevertheless, though there have been differences in assignment of categories, in practice, there remains an excess of cases with more "significant" exposures to both asbestos and nickel than controls, for all analyses though as before, the findings for nickel are significant whereas those for asbestos are borderline. Yet, if the cases with joint exposure are removed none of the differences are statistically significant.

Further analyses were conducted in an endeavour to discover whether there was confounding with other exposures particularly smoking history, alcohol consumption, other occupational fume exposures excluding asbestos and nickel, and grades of school completed and family income as indicators of socio-economic status. Although the numbers of discordant pairs when each of these factors was controlled individually becomes small, in general the risk ratios remain at a similar level and it does not appear that the increased risk associated with asbestos or nickel exposure is due to confounding by other variables.

### Conclusions

The following conclusions seemed justified from the analysis of our preliminary results and the previously reported data in the world literature.

1. Exposures of the type which may well be relevant to the etiology of laryngeal cancer are not identified through routine occupational histories. Special questioning is required in order to elicit information on such exposure. There is, however, no evidence that can be identified of recall bias in our method of questioning.
2. There has been a greater exposure to asbestos encountered occupationally in cases than controls with a risk ratio of the order of 2 to 4. This is not due to confounding by other factors with the exception of joint exposure to asbestos and nickel in three cases. The numbers available in the present analysis do not suffice to



evaluate the association of asbestos and laryngeal cancer on its own. Nevertheless, the risk ratio is of a similar order to that identified by Selikoff in his cohort studies though substantially less than that reported by Morgan and Shettigara.

3. There is a significantly greater exposure to nickel in cases than controls. This is not due to confounding except for the joint exposure between asbestos and nickel in 3 cases. This suggests an etiological role for nickel exposure and is compatible with the only other previously reported study, that of Pedersen and his colleagues.
4. The elucidation of other relevant etiological factor with regard to both asbestos and nickel exposure and cancer of the larynx requires further study and, in particular, a larger series of cases and controls. Thus, the data do not permit unravelling of the joint exposure to both agents nor is it possible at this stage from the data to determine the severity of exposure which is required to be etiologically relevant nor the appropriate latent period. On the other hand, the data currently available suggests that some of the previously identified relationships for asbestos and other cancers with, for example, a 15 to 20 year latent period and an established period of exposure over several months if not years is likely to be important.



TABLE 1  
DISTRIBUTION OF CASE/CONTROL PAIRS  
WITH RESPECT TO ASBESTOS AND NICKEL EXPOSURE  
(N.C.I.C. EPIDEMIOLOGY UNIT CODING)

		ASBESTOS			
		<u>Case</u>			
		Never	1	2	3
<u>Control</u>	Never	40	3	1	9
	Exposed*1	3	0	0	1
	Exposed 2	2	0	0	0
	Exposed 3	3	0	0	0

		NICKEL			
		<u>Case</u>			
		Never	1	2	3
<u>Control</u>	Never	53	0	1	7
	Exposed 1	0	0	0	0
	Exposed 2	0	0	0	0
	Exposed 3	1	0	0	0

\*Definition of category exposure - see text





TABLE II  
DISTRIBUTION OF CASE/CONTROL PAIRS  
WITH RESPECT TO ASBESTOS AND NICKEL EXPOSURE  
(CODING BY OCCUPATIONAL HYGIENIST)

ASBESTOS

		<u>Case</u>					
		Never	1	1-2	2	2-3	3
<u>Control</u>	Never	40	3	0	3	3	4
	1	3	0	0	0	0	0
	1-2	2	0	0	0	0	0
	2	2	0	0	0	1	0
	2-3	0	0	0	0	0	0
	3	1	0	0	0	0	0

NICKEL

		<u>Case</u>					
		Never	1	1-2	2	2-3	3
<u>Control</u>	Never	53	0	0	0	4	4
	1	0	0	0	0	0	0
	1-2	0	0	0	0	0	0
	2	0	0	0	0	0	0
	2-3	0	0	0	0	0	0
	3	1	0	0	0	0	0



TABLE III  
 DISTRIBUTION OF CASE/CONTROL PAIRS  
 WITH RESPECT TO ASBESTOS AND NICKEL EXPOSURE  
 (CODING BY OCCUPATIONAL MEDICINE EXPERT - M.D.)

ASBESTOS

		<u>Case</u>			
		Never	1	2	3
<u>Control</u>	Never	40	3	3	7
	1	4	0	0	0
	2	2	0	0	1
	3	2	0	0	0

NICKEL

		<u>Case</u>			
		Never	1	2	3
<u>Control</u>	Never	53	7	0	1
	1	1	0	0	0
	2	0	0	0	0
	3	0	0	0	0



FORMS

- 6S - Employee's Report of Occupational Disease
- 7S - Employer's Report of Occupational Disease
- 8S - Doctor's Report of Occupational Disease
- 374 - Advisory Committee on Occupational Disease
- 899 - Referral to Claims Review Branch
- 904 - Dependency Status

FORM LETTERS

- SC6 - Advisory Committee's indication of no change in award, form sent to worker
- SC7 - If indication from Advisory Committee shows change in worker's condition, form sent to worker
- SC9 - If pension award has increased, form sent to worker
- 1513
- (SC3) - Following medical examination by Board, Advisory Committee indicates no chest condition, form sent to worker

OTHER MATERIAL

- Mining History Memo
- Memo sent to Consultant for confirmation of opinion





## CLAIM NO.

[illegible]

ACCIDENT DATE

[illegible]

SOCIAL INSURANCE NO.

WEIGHT	HEIGHT	SEX	MARRIED	SPEAK ENGLISH?	OCCUPATION
FT. INS.					
NAME AND ADDRESS OF EMPLOYER WHERE YOU CLAIM YOU WERE LAST EXPOSED TO DUST.				DATE YOU FIRST ENTERED THAT EMPLOY	DATE YOU LEFT THAT EMPLOY
				IF OFF WORK NOW, GIVE DATE AND HOUR OF LAYOFF.	
				HAVE YOU PREVIOUSLY MADE A CLAIM FOR OCCUPATIONAL DISEASE	
SS					

FULL PARTICULARS OF YOUR EXPOSURE TO DUST SHOWING NAMES OF EMPLOYERS WITH DATES  
PERIOD OF EMPLOYMENT WITH EACH EMPLOYER.

IN ONTARIO			OUTSIDE ONTARIO		
EMPLOYER'S NAME	FROM	TO	EMPLOYER'S NAME	FROM	TO

**BIRTH**

**MINERS CERTIFICATE NO.**

I CERTIFY THAT ALL THE ABOVE IS TRUE AND CORRECT AND I CLAIM COMPENSATION AND/OR MEDICAL AID.



**Employee is claiming  
compensation for disability  
from industrial disease.**

MARITAL STATUS	SOCIAL INSURANCE NO.	OCCUPATION
DOEE OUR EMPLOY	MINER'S CERTIFICATE NO.	
<p>INJURED EMPLOYEE THE OWNER OR PARTNER BUSINESS OR A CONTRACTOR, SUB-CONTRACTOR, WIFE OR HUSBAND OF THE EMPLOYER? DOES DOEE HOLD THE OFFICE OF PRESIDENT, VICE- PRESIDENT, DIRECTOR, SECRETARY OR TREASURER?</p>		
<p>DOEE A RELATIVE OF THE EMPLOYER?</p>		
<p>ALLEGED THAT THE DISEASE WAS CONTRACTED DURING EMPLOYMENT OF SOME OTHER EMPLOYER?</p>		
<p>PLAIN "YES" ANSWERS ON BACK OF FORM IF NECESSARY</p>		

MAILING ADDRESS

FULL PARTICULARS INCLUDING DATES AND GRADES OF WORK OF THE EXPOSURE.

FROM	TO	GRADE OF WORK

SHOW TOTAL EARNINGS FOR 1 YEAR (52 WEEKS) IMMEDIATELY PRIOR TO THE CURRENT PAY PERIOD.		
EARNINGS	DATES OF PERIOD COVERED BY EARNINGS FROM	TO

SHOW CAUSE OF LOST TIME WITHOUT PAY			
DAYS LOST A	SICKNESS	LACK OF WORK	OTHER (SPECIFY)

DATE	OFFICIAL TITLE

SIGNATURE	PHONE NO.	<div style="text-align: center; font-size: 24pt; font-weight: bold;">7S</div> <div style="text-align: center; font-size: 10pt;">(07/78)</div>



# THE WORKMEN'S COMPENSATION BOARD, ONTARIO

100 BLOOR STREET EAST, TORONTO M4W 3C5 • TELEPHONE (416) 965-8804



## DOCTOR'S REPORT OCCUPATIONAL CHEST DISEASE

Please answer ALL questions  
fully and enclose copies of  
consultation and biopsy re-  
ports relating to this disability.

### SYMPTOMS AND COMPLAINTS (WITH DURATION)

### PAST ILLNESSES

PNEUMONIA ☐  
BRONCHITIS ☐  
RHEUMATIC FEVER ☐  
TUBERCULOSIS ☐  
DIABETES ☐  
OTHER ☐

IF POSITIVE, GIVE DETAILS AND DURATION.  
PNEUMONIA ☐  
BRONCHITIS ☐  
RHEUMATIC FEVER ☐  
TUBERCULOSIS ☐  
DIABETES ☐  
OTHER ☐

### PHYSICAL EXAMINATION — CHEST AND CARDIOVASCULAR

INSPIRATION - MAXIMUM      EXPIRATION - MINIMUM      EXPANSION - INCHES

ABNORMAL FINDINGS

BLOOD PRESSURE

HEART RATE

SIZE

RESPIRATORY SOUNDS

HEIGHT

WEIGHT

PRESENT

USUAL

### TUBERCULIN TEST

WHERE?

WHEN?

RESULT

### SPUTUM EXAMINATION

WHERE?

WHEN?

RESULT

### LUNG BIOPSY/THORACENTESIS

WHERE?

WHEN?

RESULT

### CHEST X-RAY

WHERE?

WHEN?

FINDINGS

PNEUMOCONIOSIS PRESENT?

READER

TUBERCULOSIS PRESENT?

WORKS

OSIS

EXAMINATION

SIGNATURE OF EXAMINER

8S

REV 1.76





TO: ADVISORY COMMITTEE ON OCCUPATIONAL CHEST DISEASES

WORKER FOR EXAMINATION

DATE \_\_\_\_\_ 19 \_\_\_\_\_

CLAIM NO. \_\_\_\_\_

NAME AND ADDRESS \_\_\_\_\_

OCCUPATION \_\_\_\_\_ LENGTH OF EXPOSURE \_\_\_\_\_

ATTENDING DOCTOR'S  
NAME AND ADDRESS \_\_\_\_\_

COPIES OF FORMS  
ENCLOSED.....

6S	7S	8S
----	----	----

X-RAY REPORTS	MEDICAL REPORTS
------------------	--------------------

EMPLOYER'S  
NAME AND ADDRESS \_\_\_\_\_





## Referral For Claims Review Branch

Memo No.

Claim No.

Team No.

Issue (What is being claimed and for what period.)

Objections and Opinions (If an objection or a re-opened claim, identify initiating document and its date.)

Adverse Recommendation

Authority or Policy References

Authorization

Signature — Claims Adjudicator

Date

Signature — Team Co-ordinator

Date





The  
Workmen's  
Compensation  
Board

2 Bloor Street East  
Toronto, Ontario  
M4W 3C5

Telephone  
(416) 965-8610

## Dependency Status

Claim No.

--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--

(Please print name)

residing at

(Street)

(City)

(Province)

(Postal code)

I hereby declare that:

1. I was married on the \_\_\_\_\_ day of \_\_\_\_\_ 19\_\_\_\_ at \_\_\_\_\_  
(City, town or place)

and my wife's single name was \_\_\_\_\_

2. The names and dates of birth of our dependent children under the age of 16 years, now living with us, are as follows:

Name	Date of birth	Place of birth

3. The names and dates of birth of our dependent children over the age of 16 years living with us, who are attending school or university at this date, are as follows:

Name	Date of birth	Place of birth

4. The names and dates of birth of children dependent on me, other than those already mentioned are as follows

Name	Date of birth	Present address

Please state relationship of these children to you or your wife, and why they are dependent on you \_\_\_\_\_

5. At this date, my wife and the aforementioned children are dependent for support and maintenance, and we are living together as husband and wife, and have been so living continuously since our marriage. (If any exceptions, please give details):

I hereby certify that the statements and information given above are full and true in every respect.

Signed this \_\_\_\_\_ day of \_\_\_\_\_ 19\_\_\_\_.  
Signed in the presence of:

Witness to sign here)

(Employee to sign here)

Please send your Marriage Certificate to the Board by registered mail. It will be returned to you when it has served its purpose.





2 Bloor Street East  
Toronto, Ontario  
M4W 3C3  
Telephone (416) 965-8804

**The Workmen's Compensation Board**



Dear

Claim S

Your recent examination indicates that there is no change in your industrial chest condition.

Your case will be followed closely and you will be advised if re-examination is necessary.

If you have any reasons for objecting to this decision or have any concerns or questions about the matter, please let us know as soon as possible.

When writing to us, please include both your claim number and address. To avoid delays, all address changes should be reported promptly by letter.

Yours very truly,

Claims Adjudication Branch.



2 Bloor Street East  
Toronto, Ontario  
M4W 3C3  
Telephone (416) 965-8804

**The Workmen's Compensation Board**



Dear

Claim S

Your claim for an industrial chest condition has been allowed and permanent partial disability has been assessed at        percent. You have been awarded a pension of \$        a month. This pension dates from        and will be paid so long as the disability continues.

Your case will be followed closely and you will be notified if re-examination is necessary.

If you have any reasons for objecting to this decision or have any concerns or questions about the matter, please let us know as soon as possible.

When writing to us, please include both your claim number and address. To avoid delays, all address changes should be reported promptly by letter.

Yours very truly,

Claims Adjudication Branch.



2 Bloor Street East  
Toronto, Ontario  
M4W 3C3  
Telephone (416) 965-8804

**The Workmen's Compensation Board**



Dear

Claim S

Recent examination indicates that you are entitled to a higher disability rating and this has been increased to        percent. Accordingly your pension for permanent partial disability has been increased to \$        a month, dating from        . This pension will be paid so long as the disability lasts.

Your case will be followed closely and you will be notified if re-examination is necessary.

If you have any reasons for objecting to this decision or have any concerns or questions about the matter, please let us know as soon as possible.

When writing to us, please include both your claim number and address. To avoid delays, all address should be reported promptly by letter.

Yours very truly,

Claims Adjudication Branch.





2 Bloor Street East  
Toronto, Ontario  
M4W 3C3  
Telephone (416) 965-8804

**The Workmen's Compensation Board**



Dear

Claim S

Your recent examination indicates that there is no evidence of an Industrial Chest Condition.

Your case will be followed closely and you will be advised when re-examination is necessary.

If you have any reasons for objecting to this decision or have any concerns or questions about the matter, please let us know as soon as possible.

When writing to us, please include both your claim number and address. To avoid delays, all address changes should be reported promptly by letter.

Your very truly,

Claims Adjudication Branch.



CLAIM .....

Certificate No.

MINING HISTORY

Social Insurance No.

Birth Date

Disability



DR. STEWART

( )

Examination - A/C

Note the reports on file. We intend to list for examination by the Advisory Committee.

If you agree, please make the necessary arrangements. Please also forward the enclosed copies of our reports to the Advisory Committee.





**The Workmen's Compensation Board**

2 Bloor Street East, Toronto, Ontario M4W 3C3

Telephone (416) 965-8827

**Wm. J. McCracken, M.D.**  
B.Sc., F.R.C.S.(C), M.S., F.A.C.S.  
Executive Director  
Rehabilitation Services Division

November 1976.

TO THE MEDICAL PROFESSION

Dear Doctor:

Re: Industrially Generated Disease

This letter is submitted to your attention in order to alert you about the possibilities of industrially generated disease.

As you are well-aware, we have now entered a phase in medicine where diseases, including cancers, are being identified with a causal relationship to specific natural and man-made chemicals and physical agents such as radiation.

One common denominator has become obvious. This is the lag or latency period between first exposure to risk and the onset of the disease. For instance, we have established a latency period of 10 to 15 years for asbestos dust giving rise to certain bronchogenic carcinomas, 5 to 20 years latency period for coke oven emissions to cause certain bronchogenic carcinomas, and a 20 years latency period for asbestos dust ingestion to cause certain gastrointestinal cancers.

We have asked the Ontario Cancer Foundation to give serious consideration relative to the inclusion of an occupational history with their statistics. Such information could assist in identifying cancer trends showing causal relationship.

We now ask you -- the Physicians of Ontario, to build a suspicion index into your history taking relative to these factors. In today's complex technological society the physicians, in order to evaluate the diagnostic problem, should make enquiry into occupation. This enquiry should include asking the patient if he was exposed to any chemicals, gases, radiation, noise or dust at his work place as well as at his home and in his recreational environment. Smoking, eating and drinking habits should also be included in this type of enquiry.

The development of an exposure to risk section in the functional enquiry portion of your history taking may allow you to identify the disease trend.

Finally; should you feel that there is a possible causal relationship, please advise me with identification and details. This will allow the Board to collect, collate and evaluate such reports and consider entitlement. By doing this, both the profession and the Workmen's



Compensation Board will be in a position to respond in a positive responsible fashion to such possible health threats.

This letter is submitted for your information and to solicit your co-operation in this worthwhile endeavour.

Yours very truly

WJMc\*glg

Wm. J. McCracken, M.D., F.R.C.S.(C).



May 21, 1981

Royal Commission on Matters of Health  
and Safety Arising from the Use of  
Asbestos in Ontario  
180 Dundas Street West, 22nd floor  
Toronto, Ontario  
M5G 1Z8

Dear Sirs & Madams:

I will indever to give as brief an outline on my medical history as I can and my working history up to my forced retirement in April of 1976 from the Corp. City of Sudbury.

I worked in a number of mines starting in 1937 till 1940 when I joined the army. I am submitting some dates and places but I can be out on dates as I kept no record of these dates.

I first started with Canadian Johns-Manville at there Munro mine East of Matheson Ontario in July of 1949 and left them in October of 1949 for three months returning back in January 1950 and remained with them till Feburary 1958.

I was retained as a bulldozer operator to start, then heavy duty mechanical work and fitter work of all kinds and then to mechanical foreman in all phases of mine operation. During the first eight years of this mine operation I was called upon to do a very high amount of overtime which I did not mind, as of those years I was in good health and strong.

I worked a good many seventy to eighty hour weeks and on numerous occassions I worked ~~from~~ sixteen to twenty-four hours a day on the mine property for serious equipment breakdowns. We had weather conditions in winter from 15 to 45 below zero.

It was my responsibility to see that all mine equipment was kept repaired and going and it turned out to be a never ending job. We also had the asbestos tailings dump belts and discharge equipment to maintain. Working on the tailing dump discharge was so bad at most times with blowing tailings you could hardly see or breath, using a mask was of no help as they would clog up in minutes. We all wore safety goggles as the asbestos tailings would get in our eyes and we'd be forced to come down off the dump and go to the safety room to wash out our eyes and nose.

(Cont....)





Being familiar with both surface and underground mines I was asked to take over the maintenance and the setting up of all underground equipment, hoists, underground rock crushers, pumps, rock drills, ore slushers etc. Anyone familiar with underground slushers and rock crushers can imagine the amount of dust that is raised, there's times we'd say you could almost cut it with a knife.

I might add at this point that the manager and senior officials of Munro mine done everything they could and were allowed to do to keep everything as safe as possible and keep a margin of profit, as we all understood the Company makes there profit or closes down. Unfortunately for the vast majority of us regardless of our good health, did not understand the damage asbestos dust can cause to a persons chest and lungs till its to late.

In 1961 or 62 I was called down to John-Manville plant at Asbestos Quebec for an interview for a mechanical position to work for them again but I didn't get the position but I didn't find out till years later while working for the City of Sudbury that it was on account of my chest exrays.

May I add that on trying to explain some of these working conditions to the W.C.B. on my first visit to Toronto to see them the interviewing Doctor advised me that they had all the information they needed and didn't appear to care about the enourmous amount of overtime and dust.

I will list the Doctors and diagnosis. Dr. Trobridge, 865 Regent S. South Memorial Hospital, Sudbury and others on attached list plus other pieces of information that may be of interest, if not please disregard.

On reading all the material I've recieved about the Royal Commission I can see the tremendous amount of work involved in this undertaking, I, like untold others can only hope that perhaps something may now come of this sorry mess and misery to untold hundreds of people.

I could go on and on but hopefully this might in a small way help out somehow. May you good people have patience and bear with me and my submissions, this being the first time I've ever tried to put into words my feelings and thinkings on anything of this kind.

I remain yours truly,

A handwritten signature in cursive script, reading "G.W. Cousineau". The signature is written in dark ink and is positioned above the printed name.

G.W. Cousineau



De quoi vous plaignez-vous présentement?

Asbestosis  
Bronchial Asthma, Emphysema, Pulmonary Fibrosis  
Pain in chest from coughing and sputum of blood in sputum  
Taking heavy medication four times a day for this  
B) Describe your usual daily activities.  
Que faites-vous pour occuper votre temps?  
Short walks, weather permitting,

C) Name and address of your present family doctor: How frequently do you see him? Date you last saw him?

Nom et adresse de votre médecin de famille: Le voyez-vous fréquemment? Quand l'avez-vous vu la dernière fois?

Dr. T. A. Fabres, 240 Mc Garry St. E. Marie Ontario  
June 1978 - Oct. 1978 and Dec. 1978 - This is a new doctor who  
has taken me as a patient as I no longer live in Sudbury.

D) Name and address of other doctors involved in your case over the last 2 years:

Noms et adresses des médecins qui vous ont traité durant les deux dernières années:

Dr. S. H. Meralli - 1314 La Salle Blvd. Sudbury Ont.  
Dr. S. Kivinen - 865 Regent St. Memorial Hospital Sudbury  
Dr. J. Probridge - 865 " " "

E) If you have been in hospital over the last 2 years, give name(s) of hospital, dates of admission and release, and reasons for admission(s).  
Si vous avez été hospitalisé durant les deux dernières années donnez

Admitted to Memorial Hospital  
865 Regent, Sudbury, Ont. ph. 673-84  
For complaints stated - Bronchial asthma,  
Emphysema, Pulmonary fibrosis, suspected  
Asbestos on the lungs.

Dates for two years past.  
Admitted to Memorial Hosp. Jan. 19th Released Jan. 21  
" " " " Jan. 29th 76 " Feb. 6  
" " " " Feb. 26th 76 " March 4  
" " " " March 11th 76 " March 20

Admitted to Sudbury General Hosp. March 22 " March 31

The year previous to this record 1975 I was admitted  
and released six or seven times to  
Memorial Hospital Sudbury. J. W. Cousman





NAME COUSINEAU  
NAME(S) GEORGE W  
AL ADDRESS 613 PLACID AVE  
OR TOWN SAULT STE MARIE  
AL INSURANCE NO. 1-544-708 PHONE NO. 256-8332

Never worked  
over the years

414-644-708

WEIGHT 140 HEIGHT 5 FT. 5 INS. SEX M MARRIED YES SPEAK ENGLISH YES OCCUPATION FOREMAN  
NAME AND ADDRESS OF EMPLOYER WHERE YOU CLAIM YOU WERE LAST EMPLOYED TO DUST. CAN ROCK URANIUM MINES LTD, LACIT LAKE ONT.  
DATE YOU FIRST ENTERED THAT EMPLOY FEB 17 1958 DATE YOU LEFT THAT EMPLOY OCT 1960  
IF OFF WORK NOW, GIVE DATE AND HOUR OF LAYOFF. Oct 9th 1970  
HAVE YOU PREVIOUSLY MADE A CLAIM FOR OCCUPATIONAL CHEST DISEASE NO

FULL PARTICULARS OF YOUR EXPOSURE TO DUST SHOWING NAMES OF EMPLOYERS WITH DATES PERIOD OF EMPLOYMENT WITH EACH EMPLOYER.

IN ONTARIO  
EMPLOYER'S NAME FROM TO  
CANADIAN JOHN'S JULY 1949 OCT 1949  
VILLE, A JAN 1950 Feb 15/58  
ESTOS, FIBRE  
VISION, MATHESON  
LLINGER GOLD JULY 1938 JAN 1941  
TIMMIN'S  
ECORUM MINES DEC. 1946 Sept. 1948  
ENTURE, GROUP  
R, ADDISON & Feb. 1946 Oct. 1946  
STERVILLE MINES  
CITY OF SUDBURY Jan. 11/65 April 7th 1976  
HANICAL MAINTENANCE  
OF BIRTH FOREMAN GAGNE Sept. 13 1917

OUTSIDE ONTARIO  
EMPLOYER'S NAME FROM TO  
MINERS CERTIFICATE NO. T 11250

ARE ALL THE ABOVE IS TRUE AND CORRECT AND I CLAIM COMPENSATION AND/OR MEDICAL AID.

THIS DAY OF 19 CLAIMANT SIGN HERE 68 REV 374





# United Steelworkers of America

LOCAL UNION No. 2251

AFL-CIO

IN CANADA AFFILIATED TO THE CANADIAN LABOUR CONGRESS



Telephone 942-3900

68 DENNIS STREET  
Sault Ste. Marie, Ontario  
P6A 2W9  
CANADA

Mr. Dave Adamson,  
Supervisor,  
The Workmen's Compensation Board,  
2 Bloor Street East,  
Toronto, Ontario.  
M4W 3C5

July 27, 1979

Dear Sir:

Re: Mr. George Cousineau  
613 Placid Avenue  
Sault Ste. Marie, Ontario  
Claim No. S11410055

In a letter dated February 24, 1978 the Board advised Mr. Cousineau that a recent examination indicated there was no evidence of an industrial chest condition.

In my reply to that letter dated July 18, 1978 I requested that the Board review Mr. Cousineau's claim as it had been stated by Dr. G. W. Kivinin that Mr. Cousineau definitely had a chest condition which was established as asbestosis.

Mr. Cousineau was also seen on June 8, 1979 by Dr. T. A. Ferrier of 240 McNabb Street, Sault Ste. Marie and was given the same diagnosis.

Enclosed please find a copy of Dr. Ferrier's medical opinion.

We are somewhat disturbed after reviewing Mr. Cousineau's file and reviewing the medical documentation available that the Board does not want to recognize his condition as being related to the employment or recognize that he has a chest condition.

We request that you conduct a complete review of this claim and that Mr. Cousineau be seen to determine his degree of disability as a result of his asbestosis which I am sure was not a result of walking down a city street.

Continued....



We would appreciate hearing what immediate progress is to be made in the above-noted claim.

Yours very truly,

UNITED STEELWORKERS OF AMERICA  
LOCAL UNION 2251

Dennis Abernot  
Chairman  
Compensation, Safety  
and Health Committee

DA/ec

encl.

cc - Mr. G. Cousineau ✓



# United Steelworkers of America

LOCAL UNION No. 2251

— AFL-CIO —

IN CANADA AFFILIATED TO THE CANADIAN LABOUR CONGRESS



TELEPHONE 256-6294 - 5

68 DENNIS STREET  
Sault Ste. Marie, Ontario  
P6A 2W9  
CANADA

Mr. James Vance,  
Counselling Specialist,  
The Workmen's Compensation Board,  
2 Bloor Street East,  
Toronto, Ontario.

November 17, 1977

Dear Sir:

Re: Mr. George W. Cousineau  
613 Placid Avenue  
Sault Ste. Marie, Ontario  
S.I.N. 414-644-708

Mr. Cousineau came in to see me after being in Memorial Hospital, Sudbury, Ontario and under the care of his family doctor, Dr. S. Y. Merali, who called Dr. Kivinen a chest specialist into the case. The diagnosis of Mr. Cousineau's chest condition is asbestosis.

Mr. Cousineau worked for Adventure Mines, Schumacher, Ontario from 1946 to 1949, John Mansville Munro Mine, Matheson, Ontario from 1949 to 1959, Stanrock Uranium Mine, Elliot Lake, Ontario from 1959 to 1961, Marson Construction Company, Sault Ste. Marie from 1961 to 1966 and City of Sudbury Board of Works from 1966 to 1975.6. He hasn't worked since.

We respectfully request a claim be set up to determine if Mr. Cousineau's chest condition is work-related.

Yours very truly,

UNITED STEELWORKERS OF AMERICA  
LOCAL UNION 2251

Alan MacDonald  
Chairman  
Compensation, Safety  
and Health Committee

AM/ec

cc - Mr. G. Cousineau ✓





2 Bloor Street East  
Toronto, Ontario  
M4W 3C 5  
Telephone (416) 965-8804

The Workmen's Compensation Board



February 24, 1978

Mr. George W. Cousineau  
613 Placid Avenue  
SAULT STE. MARIE, Ont.

Dear Mr. Cousineau:

Claim: S11410055


Your recent examination indicates that there is no evidence of an Industrial Chest Condition. -

Your case will be followed closely and you will be advised when re-examination is necessary.

Your address on our records is as shown in this letter. Should you change your address, please notify us promptly.

Yours very truly,

ADJUDICATION BRANCH

  
F. P. Samardak  
Claims Adjudicator  
vg



# Asbestos hazards covered up—files

WASHINGTON (AP) — An alleged industry cover-up of the danger posed to workers by asbestos is detailed in documents dating back to the 1930s, the Washington Post says.

"These files are going to be the Pentagon Papers of the asbestos industry," Barry Castleman, a consultant to the Environmental Defence Fund, was quoted as saying.

The documents include internal memoranda, files and sworn statements from several former asbestos industry officials. They are being circulated

among lawyers involved in lawsuits. The Post says.

It says claims by asbestos disease victims against industry now total more than \$2 billion.

Workers at two Southern California shipyards filed a class action lawsuit in October seeking all the profits—estimated at \$1 billion—made since 1938 by 15 of the major U.S. asbestos manufacturers.

The manufacturers are accused of conspiring to conceal the hazards of working with asbestos, dangers the companies allegedly were aware of in 1938.

"The companies made a conscious choice of profits over the health of their workers and they should not be allowed to retain those profits," said David Epstein, lawyer for the shipyard workers.

Last April, the U.S. health, education and welfare department said about 40 per cent of those who have worked with asbestos during the last 30 years may die of cancer and that about seven per cent may die of asbestosis, or "white lung disease" that can result from exposure to asbestos.

The Post account says the documents uncovered in a series of recent lawsuits

contradict industry claims that dangers of exposure were not known until 1961 when research by staffers from Mt. Sinai Hospital in New York City was published.

The Post says letters and files going back to 1934 from Johns-Manville and Raybestos-Manhattan, two of the largest asbestos firms, noted alleged efforts by senior executives to suppress information about potential harm to workers from asbestos. It says both companies deny they suppressed asbestos information.

"We have taken the position that we will be tried in the courts and not in the

press," Robert Sims, a lawyer for Raybestos-Manhattan, was quoted as saying.

"I don't think they constitute any evidence of any effort on the part of Johns-Manville to withhold relevant information or suppress the development of scientific and medical knowledge," Dennis Markussen, lawyer for Johns-Manville, said of the documents.

The Post says the industry set up research projects at a Saranac, N.Y., laboratory in the 1930s and 1940s and then prevented findings of possible asbestos dangers to humans from being published.





# Former workers in asbestos mines difficult to find

By VICTOR MALAREK

A massive search for thousands of former asbestos workers is not going very well.

In 1976, the Ontario Government and the Workmen's Compensation Board began looking for about 3,000 miners who worked at two Northern Ontario asbestos mines.

However, only 433 have been located so far, Rodney May, assistant deputy minister of the Labor Ministry's occupational health and safety division, said in an interview.

One of the mines operated by Canadian Johns-Manville Co. Ltd., the Reeves Mine in Timmins which employed about 1,000 workers, closed in 1976. The other, the Munroe Mine in Matheson, which is about 40 miles northeast of Timmins, had about 2,000 miners working there over the years. Johns-Manville closed it in 1965.

Dr. May said the search has been very difficult "because once the mines closed down, there was no other work, so many of the people moved out of the area and there is no way to get in contact with them."

He added that another major stumbling block is "there are no records avail-

able for the people who worked at the Matheson mine" and the Reeves mine had only the payroll records for the 174 miners working there when it closed.

Also in 1976, the WCB launched an extensive search for employees who worked for companies using asbestos in their manufacturing processes. But once again investigators are being hampered by poorly kept employment rolls.

William McCracken, executive director of medical services for the WCB, said search efforts are complicated by employees who have either retired or left the company for another job. Many have moved, leaving no forwarding address.

"It's a very slow, complicated process," Dr. McCracken said. "There are about 100 companies in Ontario using asbestos that we considered would constitute a risk (to workers). We've managed to get to about 30 companies so far."

Dr. McCracken said the WCB wants to find out how many of the former employees are alive or dead. "We want to find out what they died from and check the health of those who are still alive."

In both searches, the ministry and WCB have asked unions for help by checking membership records.

Dr. McCracken also said that he recently wrote a letter to the president of the Canadian Medical Association suggesting "he send a letter to all 25,000 doctors across Canada to alert them to the occupational disease problems associated with the workplace. I'm still waiting for a reply."

In addition to the searches, the Labor Ministry, which is responsible for the health and safety of workers in Ontario, has a surveillance program of workers exposed to hazardous dusts such as asbestos and silica.

It began in the late 1940s with a few hundred workers. It now encompasses thousands of employees from hundreds of companies where dust may constitute a health hazard. In 1978, 40,217 workers were examined, more than 4,100 of whom had handled asbestos.

The program, which includes X-rays and pulmonary function tests, turned up 10 new cases of asbestosis, a scarring of the lungs caused by inhaling asbestos fibre. From 1942 to 1978, 172 cases of asbestosis have been reported to the WCB.

Last year, the board awarded compensation in 13 new claims for cancer attributed to exposure to asbestos, bringing its asbestos-related cancer toll to 74 — 38 of whom also had asbestosis.

In an interview at his office, Labor Minister Robert Elgie described occupational diseases relating to exposure to asbestos as "a very serious problem."

## Indefinite term is the sentence of sex offender

By VIANNEY CARRIERE

A 49-year-old man with convictions for assaults on women dating back to 1965 was declared a dangerous sexual offender by County Court Judge E. J. Houston yesterday and sentenced to an indefinite term in penitentiary.

At the end of a four-day hearing during which he was told he was running an improper court, Judge Houston told Hugh McKinnon: "I sympathize a great deal with your problems. I have gained the impression you don't sympathize too much with mine. That is your privilege. I hope now that you will accept treatment. I have nothing but the hope all will go well for you."

The application by Crown counsel Frank Armstrong to have Mr. McKinnon declared a dangerous sexual offender was complicated by Mr. McKinnon's refusal to co-operate with psychiatrists assigned to examine him and report on his condition.

Mr. McKinnon was convicted by Judge Houston last September of severely beating his 86-year-old mother.

Judge Houston was told that McKinnon had a long history of violence against





...story of violence against  
...a very serious problem.  
However, Dr. Elgie  
stressed that many of the  
industrial health and safety  
problems of the past will be  
corrected by Bill 70 — an  
Act Respecting the Occupa-  
tional Health and Occupa-  
tional Safety of Workers.  
"Bill 70 will have a major  
input for workers in terms of  
what risks are involved in  
the work they do," the min-  
ister said.  
Dr. Elgie said that under  
the bill, employers will have  
to inform workers of what  
risks they might be exposed  
to. "But the greatest hope  
lies in health and safety  
committees," he noted.  
These joint management-  
labor committees are man-  
datory in all workplaces  
with 20 or more employees.  
(There are some exceptions  
such as theatres, stores,  
museums, offices, libraries  
and hotels.) They will identi-  
fy situations that may be a  
source of danger to workers  
and make recommendations  
to the employer on correct-  
ing them.





FLOYD/SHIRLEY LEFEBVRE  
13 OLD ORCHARD AVE  
CORNWALL ONT  
K6H 2H1

WC  
71

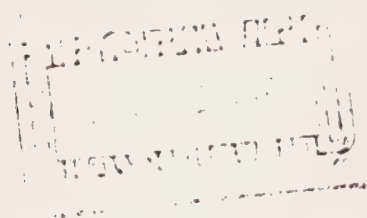
MR LINDA

JUNE 8/71

I READ YOUR NOTICE IN OUR LOCAL PAPER. I  
DID NOT ONLY LIKE A COPY I WOULD LIKE TO  
BE INVOLVED.

I WORKED FOR DOM TAR CONSTRUCTION MATERIALS  
CORNWALL AS A BEATER OPERATOR AND AS A  
HELPER HANDLING ASBESTOS FOR QUITE SOME  
TIME PRIOR TO 1972. WHEN PIPE BUSINESS  
CEASED OPERATIONS

I HAVE KEPT A LIST OF OPERATORS THAT  
HAVE PASSED AWAY PRIOR TO RETIREMENT  
ALSO. THE EMPLOYEES WHO HAVE A RESPIRATORY  
PROBLEM ALONG WITH MYSELF.



THANK YOU  
Floyd Lefebvre

FLOYD/SHIRLEY LEFEBVRE  
13 OLD ORCHARD AVE  
CORNWALL ONT  
K6H 2H1



WC  
72

Pickering, Ontario  
June 27, 1980

Dr. Stefan Dupre, Chairman  
Royal Commission on Asbestos

Dear Sir--

As a 30 year asbestos worker, there is one facet of the problem that I feel should be investigated.

On two visits for tests at the building on Grosvenor Street (corner of Bay), at the request of the Workmen's Compensation Board, during tests, I was asked to breathe various gas compounds. During the tests I felt really good and passed all tests with flying colours. Later in the day, and the next couple of days, I could hardly breathe. I thought it was my imagination until I noticed the same reaction in some fellow workers. Then, last winter with the media revelation that the W.C.B. had used truth drugs on the victims without the victim's knowledge or consent, I began to wonder, to the extent that I refused to go for tests last winter.

I think your Commission should investigate what these gases are, and their effect on human beings.

R.B. Diamond  
1987 Glendale Drive  
Pickering, Ontario  
L1V 1V8

(no phone calls please, as there is an illness in the house)





WC  
73

SEP 28

24 Fenwood Heights,

Scarborough, Ontario

September 22, 1981

Gentlemen Of The Royal Commission,

My name is Betty Glaser. I am writing on behalf of my husband Gustav Glaser. I was at The Royal Commission in the spring. At that time I explained that my husband is a victim of asbestosis. My husband has appealed his case to The Workmen's Compensation Board for complete disability. The hearing took place April 22, 1981. To date we have not heard from The Appeal Board.

During the summer I have been in touch with our M.P. Mr. F. Drea, Mr. R. G. Elgie and Mr. Lincoln Alexander. I am enclosing copies of letters from Mr. L. Alexander. I have asked them why such a lengthy delay. They have assured me that after receiving our solicitor's submission The Appeal Board will arrive at a decision. I know that Gus is not the only person awaiting their decision but, five months seems a long time.

I want to thank Mr. J. S. Dupre for forwarding my brief to Mr. L. Alexander. I am glad that finally because of The Royal Commission the ordinary person like myself can tell their story of how badly we have been treated by The Workmen's Compensation Board. My hope is that when all the evidence is gathered there will be many changes made in The Workmen's Compensation Board.

I thank you very much for listening to me. It helps to know that there is justice in the end.

Your truly,

*Betty Glaser*





## The Workmen's Compensation Board

2 Bloor Street East, Toronto, Ontario M4W 3C3

Telephone (416) 965-8880

Lincoln M. Alexander, Q.C.  
Chairman



July 20th, 1981

Mrs. E. Glaser,  
24 Fenwood Heights,  
Scarborough, Ontario.  
M1N 2V7

Dear Mrs. Glaser:

Re: Claim S8533251 - Mr. G. Glaser

As you were informed by Dr. Dupre, a copy of the transcript of your testimony before the Royal Commission on Matters of Health and Safety arising from The Use of Asbestos in Ontario was sent to me.

You expressed a number of concerns pertaining to the handling of your husband's claim by the Workmen's Compensation Board.

The matter of the level of permanent disability assessment of Mr. Glaser's compensable lung condition is still before the Appeal Board and it would not be proper for me to comment on this aspect.

You questioned the delay, following the hearing, without a conclusion by the Appeal Board. In your testimony before the Royal Commission, you stated you were told at the Appeal Board hearing that there would be a decision some time in July. A review of the transcript of the Appeal Board hearing does not confirm that you were given any date or time limit for a decision.

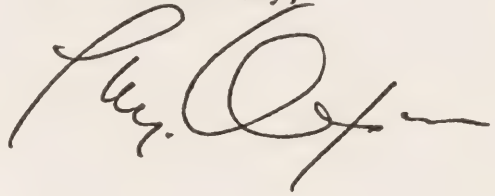
The Appeal Board makes every effort to conclude on appeals as soon as possible following a hearing where the documentation on file is complete. This was not possible in your husband's case. You informed the Appeal Board that your husband had undergone testing a few weeks previous to the Appeal Board hearing by the Advisory Committee on Occupational Chest Diseases. To ensure the Appeal Board had



all the evidence to arrive at a fair decision, it was necessary to await a report from the Committee and subsequently, to obtain further comment and opinion from the Board's Consultant in Chest Diseases. This additional documentation has been made available to your husband's representative, Mr. D. Ublansky, who has informed us he will make further submissions for the Appeal Board to consider by the end of August 1981.

While I appreciate the concern which you and your husband have in awaiting a decision, please be assured the Appeal Board, after receiving your solicitor's submission, will conclude as quickly as possible.

Yours sincerely,

A handwritten signature in dark ink, appearing to read 'P. D. Ublansky', with a long horizontal flourish extending to the right.

LMA/hh

c.c. - Royal Commission  
Mr. Ublansky







**The Workmen's Compensation Board**

2 Bloor Street East, Toronto, Ontario M4W 3C3

Lincoln M. Alexander, Q.C.  
Chairman

Telephone (416) 965-8880



4 September 1981

Mrs. E. Glaser,  
24 Fenwood Heights,  
Scarborough, Ontario.

Dear Mrs. Glaser,

Re: Claim No. S-8533251

I have received your letter outlining your very serious concerns, and it is unfortunate that the mail strike delayed receipt of my letter to you.

Please be assured that every consideration will be given to your husband's case but the decision is, of course, governed always by the terms of the Workmen's Compensation Act.

Your courtesy in writing to me is very much appreciated.

Yours sincerely,





# The Workmen's Compensation Board

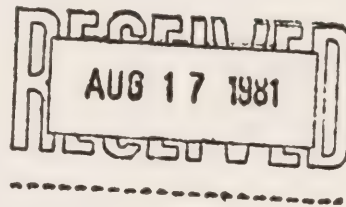
2 Bloor Street East, Toronto, Ontario M4W 3C3

Telephone (416) 965-8880

Lincoln M. Alexander, Q.C.  
Chairman



7 July 1981



Ms. Linda Kahn,  
Executive Co-ordinator,  
Royal Commission on Matters of Health  
and Safety Arising from the Use of  
Asbestos in Ontario,  
180 Dundas Street West, 22nd floor,  
Toronto, Ontario. M5G 1Z8

Dear Ms. Kahn,

Re: Royal Commission on Asbestos  
Public Hearing, Monday, June 8, 1981

Thank you very much for your letter under date of June 25th, with enclosure as stated, being the transcript of the evidence related to Mrs. Betty Glaser, taken at the time of her presentation to the Royal Commission on Asbestos on Monday, June 8th.

I have read the entire document and I am pleased that you have given me an opportunity to knowing Mrs. Glaser's concerns.

Yours sincerely,

Royal Commission on Asbestos  
Copy For Your Information

To: \_\_\_\_\_

From: \_\_\_\_\_





**The Workmen's Compensation Board**  
2 Bloor Street East, Toronto, Ontario M4W 3C3

Lincoln M. Alexander, Q.C.  
Chairman

Telephone (416) 965-8880



July 20th, 1981

Ms. Linda Kahn,  
Executive Co-Ordinator,  
Royal Commission on Matters of  
Health and Safety Arising from  
the Use of Asbestos in Ontario,  
180 Dundas Street West,  
Twenty-Second Floor,  
Toronto, Ontario.  
M5G 1Z8

Dear Ms. Kahn:

Re: Claim S8533251 - Mr. G. Glaser

Thank you for your letter of June 25th, 1981  
enclosing a transcript of Mrs. Glaser's presentation to  
The Royal Commission on Asbestos.

I have written to Mrs. Glaser regarding the concerns  
which she expressed during her presentation and enclose a  
copy for your interest.

Yours sincerely,

LMA/hh  
Encl.





July 20th, 1961

Mrs. E. Glaser,  
24 Fenwood Heights,  
Scarborough, Ontario.  
MLN 2V7

Dear Mrs. Glaser:

Re: Claim S8533251 - Mr. G. Glaser

As you were informed by Dr. Dupre, a copy of the transcript of your testimony before the Royal Commission on Matters of Health and Safety arising from The Use of Asbestos in Ontario was sent to me.

You expressed a number of concerns pertaining to the handling of your husband's claim by the Workmen's Compensation Board.

The matter of the level of permanent disability assessment of Mr. Glaser's compensable lung condition is still before the Appeal Board and it would not be proper for me to comment on this aspect.

You questioned the delay, following the hearing, without a conclusion by the Appeal Board. In your testimony before the Royal Commission, you stated you were told at the Appeal Board hearing that there would be a decision some time in July. A review of the transcript of the Appeal Board hearing does not confirm that you were given any date or time limit for a decision.

The Appeal Board makes every effort to conclude on appeals as soon as possible following a hearing where the documentation on file is complete. This was not possible in your husband's case. You informed the Appeal Board that your husband had undergone testing a few weeks previous to the Appeal Board hearing by the Advisory Committee on Occupational Chest Diseases. To ensure the Appeal Board had

...../2



all the evidence to arrive at a fair decision, it was necessary to await a report from the Committee and subsequently, to obtain further comment and opinion from the Board's Consultant in Chest Diseases. This additional documentation has been made available to your husband's representative, Mr. D. Ublansky, who has informed us he will make further submissions for the Appeal Board to consider by the end of August 1961.

While I appreciate the concern which you and your husband have in awaiting a decision, please be assured the Appeal Board, after receiving your solicitor's submission, will conclude as quickly as possible.

Yours sincerely,

LMA/hh

c.c. - Royal Commission  
Mr. Ublansky



SEP 28 1981

Dwight Oland  
14 Roosevelt Ave.  
Ajax, Ontario

Sept. 23, 1981

Dear Linda:

Enclosed please find a copy of "Summary of Information" as compiled by the W.C.B., and a copy of my appeal of the W.C.B. decisions.

The hearing was held April 1st, 1981, and I was subsequently examined at 50 Grosvenor Street on May 13 last. Dr. Vingilis seemed to act somewhat reserved at this interview - however, I make note of the fact that he thought the x-ray plates weren't quite good enough and said I would have to have two more taken. He changed his mind when I told him I had already had one set that was thrown out - or, I should say, I was twice exposed to x-rays sans x-ray plates! and then I was x-rayed twice more.

I was told I would be given a decision in July - but now it is the end of September and no word yet.

I will forward a copy of their decision when I receive it.

Thank you for your indulgence and consideration.

Yours truly,

Dwight Oland

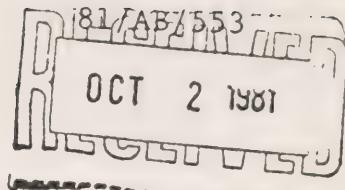
2 enclosures.





THE WORKMEN'S COMPENSATION BOARD

An appeal by Mr. Dwight Oland,  
Claim S10458096, against the  
Decision of the Claims Review  
Branch dated January 9, 1980.



COPY

APPEAL BOARD

DECISION

On April 1, 1981 the Appeal Board heard an appeal by Mr. D. Oland, who was represented by Mr. D. Ublansky, Union Representative. Mr. K. Montgomery, Union Representative, attended as an observer.

Mr. Oland claimed that the 10 per cent permanent partial disability award did not adequately represent the degree of residual occupationally induced asbestosis.

Mr. Oland has entitlement for asbestosis following exposure to asbestos while in the employment of Canadian Johns-Manville. The condition was diagnosed in August 1979, and a 10 per cent permanent partial disability award was granted in recognition of the residual disability.

Mr. Ublansky outlined the nature of Mr. Oland's disability, and emphasized that the condition was not accurately diagnosed until 1979, although Mr. Oland suffered from asbestosis for some time. He contended that the symptomatology, the effects of the disability on Mr. Oland's life style, and the lack of earning capacity, should be considered when determining the permanent partial disability award. Mr. Ublansky argued that the arthritic condition did not significantly disable Mr. Oland and that the compensable condition prevents him from returning to work.

Mr. Oland submitted a brief dated April 1, 1981, which was discussed at length.

The Appeal Board directed that a report from the Advisory Committee on Occupational Chest Diseases be secured, and that the matter be reviewed by the Board's Medical Branch. Mr. Ublansky informed the Board on August 20, 1981 that he had no further submissions to make.

The Appeal Board has considered the presentations made at the Hearing and the evidence on record.

The Appeal Board notes and accepts:

- that medical evidence does not establish the presence of pulmonary asbestosis until August 23, 1979.

Continued---



Claim: S10458096 - Mr. Dwight Oland

Page 2

- the opinion of the Advisory Committee on Occupational Chest Diseases, as indicated in the report dated May 29, 1981, that Mr. Oland experiences slight asbestosis, active rheumatoid arthritis, and that the degree of disability had not changed.
- the opinion of the Board's Medical Branch that medical evidence does not establish a disability greater than recognized by the permanent partial disability award of 10 per cent.

The Appeal Board concludes, therefore, that the 10 per cent permanent partial disability award properly reflects the degree of residual chest disability and that the award is correctly calculated and dated from August 23, 1979.

The appeal is, therefore, denied.

DATED, at Toronto, Ontario, September 15, 1981.

DECISION OF APPEAL BOARD  
PURSUANT TO SECTION 76 OF THE  
WORKMEN'S COMPENSATION ACT

*R. Farguherson*  
Registrar of Appeals





Mr. Chairman, and Commissioners -

If it please the Board, I would present a brief on my behalf to show cause for this appeal.

I have some notes with which I hope to clarify my position leading up to, and following the submission of my claim for compensation.

Also I have chronologically compiled a file of correspondence from which I propose to summarize various findings and impressions relating to my chest condition.

I have formulated comments with which to debate the contents of the Summary of Information that was forwarded to my Union Legal Counsellor, Mr. Dan Ublansky.

In 1975 while being examined by the attendants of the mobile X-ray unit at Johns-Mansville, I asked one of the attendants what the figures in the XR column meant. He told me that as long as it was a zero or a one (1) there was nothing to worry about, but if it got up to a 3 or a 4, you definitely had asbestosis.

My card read 2 for 1972, 3 in 1973 and 4 in 1974.

The job I had required very little physical effort, and I was not aware of any drastic change in my breathing, or chest condition while at work, but at other times when I played a game of badminton - or ran around a baseball diamond, or shovelled snow, I got short of breath and my heart would pound for a while; also I might start coughing for no apparent reason - just a dry sensation in my throat.

I really didn't attribute such discomfort to any chest disease. I thought it was because I was approaching the ripe old age of 60 - but just the same, I was still able to do a number of push-ups.

Even so, I was alarmed at what was on my file because it indicated I was asbestotic. I began to disbelieve what the Doctors at 50 Grosvenor St. kept telling me - that I was in A-1 shape - with no asbestosis. I decided I would like to get away from J.-M. and work elsewhere.

Although I was aware of being at that age, I wasn't going to find a job that paid as well, but I was willing to try - if I could get some help from the Workmen's Compensation Board.

My claim dated Sep. 23, 1975 was denied. However, I was invited to attend a session conducted by Dr. Stewart on June 14/76.

During my interview he asked a couple times if I coughed - and I told him no. I didn't attribute coughing to anything besides smoking too much - and I didn't smoke, or having a head or chest cold - and I rarely had one of those. I didn't connect it with the sort of dry cough that came and went with overtaxed breathing following exertion, or as when I spent any time in a dusty area - or when I went out into the cold air.





My Checker's job kept me in the office quite a bit of the time, also I was out of the building a good deal which perhaps tended to create a false sense of security - because previous to the checker's job, I worked on the floor for 14 years.

I was asked if I would consider rehabilitation, but I was undecided just then, and wanted to learn more about it. Later I asked the union president to get me additional information about Rehab., and by the time my name was put on the list, things seemed to have taken on the dead letter aspect - delayed by the people in the Rehab. branch as far as I could find out.

It was just about the same time, in the fall of 1976 I think, that one of the Company Doctors interviewed all men who had worked there 20 years or so, and spoke to them individually in confidence in his office.

When I went in he talked about my chest condition, reports of my X-rays, and my examinations. He asked if I had put in a claim for compensation. I told him I had, but it wasn't accepted. He then told me I should resubmit, or appeal. He said I shouldn't be working there according to my medical report.

I didn't know his name, and I didn't see him around afterwards: but then I wasn't seeing any of the company doctors anyway - I was getting my check-ups from my family doctor.

My Claim No. S10458096 was denied on Dec. 16/75 because quote "no significant disease noted".

I suggest the Claims Review Branch was not sufficiently and accurately supplied with the full impact of all the examinations, findings, and impressions as compiled from the date of the first accepted admission of signs of asbestosis showing up in 1967.

I wish now to expound a resume of my chest conditions compiled from various sources of information.

According to Company Health Progress Notes -

from 1953 to 1956 the results of tests were negative, and in 1958 essentially negative. Then in 1966 they were recorded as essentially within normal limits - traversing from a distinguishable condition of negation to a marked condition of "within normal limits".

Question: what is, or how does one describe normal---

--- what are the parameters of normalcy---

In 1967 - company health report shows calcification - a condition which is no longer normal.

In 1968 X-ray 504257 - "asbestos showing up".

The 1970 file card contained the numeral 2 in the XR column and in 1973 rose to a 3.

Correspondence:

May 25/73 - X-ray 727965---slight increase in density, and pleuresy noted - Dr. Vingilis.

May 31/73 - restriction of ventilation - Dr. Budlovski.

June 27/73 - obstructive airway disease - Dr. Khamsi.

Dec.7/73 - X-ray 671459 - increased markings - Co. file.

May - 1974 - my mobile file card read 4 in the X-R column.

June 17 & 18/74 - X-ray 175948--- pleural thickening, and

.../3



some obstructive ventilative defect (although a non-smoker for 7 years) - Ajax Hospital  
Feb. 2 & 12/75 - X-ray 187733--- more changes following previous X-rays in 1974---- calcification----- obstructive airway disease - Ajax Hosp.

April 22/75 - Correspondence to Dr. McIlveen from the Ministry of Health states "essentially negative until 1973", but previous records show that it was no longer "essentially negative" in 1966 - then the description was "within normal limits", and progressed to calcification in 1967 - then in 1968 asbestos showed up, and so on to when in 1970 a 2 appeared in the XR column followed by a 3 in 1973. This correspondence is not compatible to reports of 1966.

April 22/75 - Dr. Vingilis sent some information I requested - X-Ray film, and copies of my records to Dr. McIlveen. Included was what was thought to have been a true copy of my green mobile Unit record card - the card which accompanies the Unit on its regular visits - but this was not the case. A substitute card was made up and photocopied, but it did not contain some information for 1973 and there was none for 1974.

I cannot help but question why a copy of the original card was not sent -- it would have been easier than writing out another card, then making a photocopy anyway: the original had to be available to report even some of the included information.

Correspondence from the Ministry of Health refers to X-Ray L832162 of June/75 which is indicative of additional changes.

Dec 22/75 - X-Ray 850537 again showed pleural thickening--- company files.

Jan.21/76 - A report on X-ray film quotes further changes were observed - since June/75 - Ministry of Health.

June/76 - The company file reports that the opinion was pleural fibrosis due to asbestos dust.

June 7/76 - The Rehabilitation Division stated I had been identified as showing effects of asbestos.

Sept.14/76- The Team Co-ordinator of the Adjudication Branch stated my records had been reviewed - and that I did not have asbestosis sufficient to allow my claim. It would be interesting to know if ALL my records were made available to them, and what kinds of information was studied

Speaking of insufficient asbestosis - What is sufficient - Who can answer -- How would he prove I was non-compensable before 1975?





- Jan.7/77 - X-ray (of Nov.76) 860855 - again pleural thickening mentioned, and pleural fibrosis due to asbestos dust.
- Feb.18/77 - A lung scan by Dr. Khamsi revealed further changes since November, 1976. Dr. Khamsi implied that exposure to asbestos may well have contributed to bronchitis and lung damage, and he inferred some compensation could be considered.
- July 17/78- Communication from the Dept. of Labour to my family doctor revealed further changes since the examination in 1977, and again "early asbestosis" was concluded ----

I would ask what does early asbestosis mean--- considering it began showing up in 1967 and 1968 - ten or eleven years earlier--- Do different people have their own idea or criterion to use--- At what turning point does it change from non-compensable to compensable ---

I recall Dr. Budlovsky's telling me that it sometimes takes ten to fifteen years of exposure to congest a sufficient amount of asbestos dust to indicate asbestosis, and that once it showed up, there is no cure or chance of recovery, that the condition can only change for the worse --

Considering the reports since 1966, I can't help but wonder how, after eleven more years of dust accumulation revealed in the numerous references indicating many successive slight changes and increases, that such summation can not substantiate other than so called early asbestosis.

In August, 1979 I was again examined at 50 Grosvenor Street, Toronto. After the several tests Dr. Vingilis asked me to join him in his office, and before I left to go home, I thanked him for putting my mind at ease.

He told me that although I had some slight asbestotic effects, I should have no fear of ever becoming compensable. He elaborated convincingly displaying several X-ray plates, showing me what he said was taking place in my lungs from year to year. I was elated, and told him that it was the best news I'd heard in a long time.

My elation was short lived. In December I received a compensation cheque retroactive to the day Dr. Vingilis told me I was O.K.

It was then I thought perhaps Dr. Vingilis must have made a mistake in August - and I began to wonder if he'd made any other mistakes since it had first been recognized I had asbestosis showing up.

In keeping with my apprehension, I contacted the Union President who in turn informed the Claims Adjudication Branch of our objection to their decision in granting 10% disability.

The objection was subsequently denied, and an appeal was instituted.

I felt there may have been errors or discrepancies in the information contained in my medical files which were placed at the disposal of the Advisory Committee and the Review Branch. Also I began to search for additional substantiative evidence.





Dr. Gabrielle received communication from the Ministry of Labour as to further changes found on X-ray L 949144.

I phoned Dr. Vingilis on April 11/80 and asked to have my X-ray films and medical records forwarded, particularly asking again for a copy of the specific file card previously mentioned, and he agreed to do so.

A letter from Dr. Vingilis dated April 14/80 informed me the films and records as requested had been forwarded, but when I visited Dr. Gabrielle I found that at least 3 X-ray films were missing - numbers 850537, 867842 and 949144. Neither did he receive the copy of my mobile unit file card. I'm not sure about the X-ray plates as to why they weren't sent, but I know the file card was available.

Dr. A. T. Salmon had me X-rayed at the Ajax Hospital on April 17/80, and a report was received with regards to X-ray 292075, dated April 17/80 from Dr. Lillian Hsu. Perhaps such report is already in my file.

April 28/80. Correspondence to Dr. Gabrielle from the Ministry of Labour omits reference to my tests of August, 1979 and neglects to mention the pension award.

A report from Dr. Salmon dated August 22, 1980 refers to the X-ray of April 17th. He inferred there were further changes in the lungs, and that the X-rays have been suggestive of asbestosis since 1973. He advocated having my claim reviewed.

On August 25th, 1980, at 50 Grosvenor Street, Dr. Vingilis gave me a card that showed results of tests alleged to have transpired at the plant in July, 1980 with the mobile unit. I was unable to perform any such test at that time - and naturally I was indignant at seeing results of the tests portrayed on the card.

I asked the doctor how they arrived at the figures shown since there had been no test, and he said something like - I guess they just took an average of how things had been going, and put down figures to agree with such assumption. I wonder how many other reports were compiled and published after the same fashion---

It seems apparent that erroneous implications were suggestively responsible as to why the Claims Adjudication Branch advised me on September 17, 1980 that my award of 10% had been confirmed following my being examined on August 25th, 1980. The Advisory Committee reported there was no change in my classification as a silicotic. I can appreciate that statement, but why should such a report be issued --- I don't happen to have silicosis - at least so I have been told, but I am being compensated for asbestosis. My doctor doesn't have a copy of that medical examination of August 25/80 at 50 Grosvenor Street.



Over the past 4 or 5 years it has become increasingly more difficult for me to persue much physical effort. My shortness of breath has curtailed ordinary house chores and endeavours such as cutting the grass, trying to shovel snow, sweeping snow off the car, cutting the hedge, and raking up leaves. Other aspects of my life have been affected as well - swimming, hiking, playing a game of badminton, running the bases while playing ball - even dancing after a few minutes is too much for me.

At these times my chest seems to tighten up more, and breathing becomes an effort - I sometimes find myself taking quick shallow breaths, and that my chest feels tight and dry. It frightens me when it hurts a little to breathe deeply, and when I start to cough a dry cough with nothing to cough up. My chest feels like a dried out football that is hard to blow up - not so much give anymore.

I get the same feeling when I go up and down stairs, or after walking a block or so to my daughter's house.

Such was the state when I lost my job. As you know, Johns-Manville closed down the department in which I worked for 30 years - Transite Pipe.

If this had not have happened, I would have continued working - because my job as Quality Control Checker required very little physical work and I was doing O.K.

I was forced to work elsewhere in the plant. I tried a clerical type of job in the maintainence department, but for reasons best known by the Company, my training on the new job was insufficient for me to hold it. The Company told me I would have to bump into the Shipping Dept. where I would have to manhandle bales of fibreglass, piling them into dusty trucks.

I told them I wouldn't be able to do that kind of work very long before getting short of breath and breaking out into a coughing spell. They said they had nothing else to offer me --- after 30 years --- not even in a non-union job. That was it, they said, so I applied for sick leave.

As has been reported, I was afflicted with arthritis; and at one time was on sick leave because of my knee commencing on the 27th of March, 1974. That was the only time - up until 1980 - that arthritis prevented me from working. I believed at the time I was offered the Shipping job, that each, or either of my conditions, arthritis and asbestosis would have proved prohibitive in that job. This was corroborated by Dr. Gabrielle's letter of August 7, 1980.

I had the option of claiming compensation for asbestosis, or applying for Travellers Insurance benifits. Because, as you are aware, I was waiting for the Union President to imminently ask for a new appeal date, at which time my claim would be studied anyway, I chose to accept Insurance benefits in the interim.

After that the Union changed presidents, and no one worked on my appeal from that direction until Mr. Ublansky requested a Summary of Information on December 3, 1980.

A letter signed by Dr. Gabrielle, dated Feb. 17/81 lends credence in the need for the initial submission of my claim in 1975, and subsequent appeal of following Decisions.

.... /7





DEBATE OF SUMMARY OF INFORMATION TO JAN. 9, 1980.

In perusal of the Summary of Information forwarded to Mr. Ublansky I found the contents implicative rather than conclusive. I found irrelevant quotes with regard to arthritis which were used and displayed in such a manner as to obscure and detract from the true object and purpose of the Summary, namely the history and affects of asbestos contamination.

Statements with regard to arthritis were assumed and implied, taken out of chronological context, and in some instances false.

This proffered Summary appears to have been compiled with the intention to mislead and confuse for the sole purpose of undermining the applicant, and to show disregard for a valid claim.

Many yearly examination findings and reports seem to have been glossed over, or condensed, and reiterated to produce a Summary of minimal consequence.

Page 1. Reference to the inability to work in the alleged physicians's report in 1975 is false. Pay cheque stubs account for 52 weeks for that year, as being employed.

Page 2. What can be extended to support quote "it was felt" regarding the permanence of inability to work:- and (para. # 1) also whether it pertained to arthritis or asbestosis. Again, The statement that the applicant stopped working in 1967 is false. The only lost time was due to a legal strike.

Page 4. Perhaps someone will come forward to prove that 27 line years of continuous working in asbestos dust was not 13/14 responsible for the lung condition. There is no history of contracting any other lung disease, or exposure to other contaminants elsewhere.

(para. 6) The statement pertaining to 10 years of arthritis prior to the 1977 report is false. Also malicious is the reference to arthritis in all joints, and grossly limited activities due to arthritis.

Previous to 1979 the claimant had not been affected or examined for arthritis in the alleged areas.

Page 6. It could but be expected that the Advisory Committee would reiterate Dr. Vingilis' explanatory 1980 allegations. (para. 9) Obviously he neglected to say that during my conversation with him in his office he was adamant and provocative in that he refused to reply to questions about the retroactive compensation award; that he first denied having, but later showed me an almost exact DUPLICATE of the mobile unit file card. Neither, I suppose, did he mention I was incited at being shown a written report of an examination in July, 1980 at the plant - I had no such examination.

He tried to create the allusion that I was "unhappy" because of arthritis, but it appears that the doctor has difficulty in differentiating between discontent and irritation - and he used arthritis as a subterfuge.

I have sundry file correspondence relating to many of the points set forth in my brief that I would be willing to share with the Board.





(from the W.C.B.)

SUMMARY OF INFORMATION

Claim: S10458096 - Dwight Oland

ISSUE:

Mr. Oland is requesting an increase in his 10% permanent partial disability award.

DIAGNOSIS:

Entitlement:

Asbestosis

HISTORY:

On May 26, 1950, Mr. Oland commenced employment with Canadian Johns Manville. For approximately 25½ years he worked in the Transit Department. In September of 1975 while still employed by Canadian Johns Manville as a production checker Mr. Oland made a claim for asbestosis. A report was received from the attending physician showing a diagnosis of chronic obstructive lung disease. The report indicated that Mr. Oland was also suffering from arthritis and was unable to work. In November of 1975 the chest x-ray report from the Department of Health was reviewed by the Consultant in Chest Diseases. No significant disease was noted at that time and based on that report the recommendation was made that entitlement not be accepted. On December 16, 1975 the Claims Review Branch denied entitlement in Mr. Oland's claim because there was no evidence of silicosis or any other compensable chest condition.

In February of 1977 Mr. Oland was examined by a Specialist in Internal Medicine with respect to evaluation of his lungs for asbestosis. A lung scan revealed only border line changes. Respiratory function tests showed moderate obstructive airway disease.

On August 8, 1977 Mr. Oland was examined by the Advisory Committee on Occupational Chest Disease. At that time it was felt that there was not enough evidence to warrant a diagnosis of pulmonary asbestosis and a recommendation was made that Mr. Oland's claim not be allowed.

Mr. Oland was re-examined by the Advisory Committee on Occupational Chest Disease August 23, 1979. At that time a diagnosis was made of a slight pleural and parenchyma asbestosis. A disability rating of 10% was recommended.



Claim: S10458096 - Dwight Oland

Mr. Oland's Union appealed the 10% permanent partial disability award on Mr. Oland's behalf. On January 9, 1980 the Claims Review Branch concluded that the 10% permanent partial disability award granted to Mr. Oland was in keeping with the degree of disability residual to the exposure to asbestos. The Union's objection to the amount of the permanent partial disability award was denied.

DOCUMENTS:

1. Initial Report of Attending Physician, September, 1975:

(2) The physician advised that Mr. Oland was suffering from chronic obstructive lung disease. He also had arthritis. (2) It was felt that Mr. Oland would be permanently disabled from physical work. Mr. Oland had stopped working in 1967. (5) X-rays and pulmonary function tests were recommended. (3)

2. Mr. Oland's Report of Occupational Chest Disease, October, 1975:

Mr. Oland advised that he had begun working for the accident employer on May 26th, 1950. His occupation had been that of a production checker. Mr. Oland indicated that he had not yet laid off work. He stated that he had worked in the Transit Department for 25½ years in different areas. In the Transit area products were formed, machined, packaged and stored. Mr. Oland had also worked in the crushing area where the air had been dust laden.

3. Employer's Report of Occupational Chest Disease, October, 1975:

With their report the employer included Mr. Oland work history. The employment records revealed that from July to September, 1949 Mr. Oland had worked as a casual labourer. From September to December of 1949 he had been a stander and from December of 1949 to May of 1950 he had worked in a laboratory. Mr. Oland had been hired by Canadian Johns Manville on May 26th, 1950. He had been a general worker working with asbestos, cement and silica up until September 14th, 1952. From September 15th, 1952 until March 1st, 1953 Mr. Oland worked as a spare with asbestos, cement and silica. He was transferred March 2nd, 1953 but worked with asbestos, cement and silica up until August 12th, 1956. He was re-classified from September 13th, 1956 until June 16th, 1957

-continued-





and worked as a checker in shipping. Mr. Oland still worked with asbestos, cement and silica. From June 17th, 1957 until November 29th, 1959 Mr. Oland worked in salvage with asbestos, cement and silica. He was a sample lathe operator from November 30th, 1959 until August 5th, 1962. Mr. Oland was absent with a right ankle injury from August 6th, 1962 until October 15th, 1962. He was re-instated October 16th, 1962 and worked as a sample lathe operator up until March 6th, 1963. Mr. Oland was ill from March 7th, 1963 until March 18th, 1963. He was reinstated March 19th, 1963 and worked up until September 12th, 1963 as a sample lathe operator with asbestos, cement and silica. Mr. Oland was ill from September 13th, 1963 until September 16th, 1963. He was reinstated September 17th, 1963 and worked as a sample lathe operator until September 22nd, 1963. Mr. Oland was ill again from September 23rd, 1963 until October 9th, 1963 and was reinstated October 10th, 1963. He then worked as a sample lathe operator until May 10th, 1964. From May 11th, 1964 until October 11th, 1970 Mr. Oland was a production checker working with asbestos, cement and silica. He was ill with a coronary from October 12th, 1970 until February 15th, 1971. Mr. Oland was reinstated as a production checker from February 16th, 1971 until April 26th, 1972. Mr. Oland was ill with the flu from April 27th, 1972 until May 1st, 1972. He was reinstated as a production checker from May 2nd, 1972 until December 27th, 1972. Mr. Oland was ill with the flu again from December 28th, 1972 until January 22nd, 1973. From January 23rd, 1973 until March 27th, 1974 Mr. Oland worked as a production checker with asbestos, cement and silica. He was ill with arthritis from March 28th, 1974 until September 3rd, 1974. Mr. Oland was reinstated on September 4th, 1974 and worked as a production checker up until April 10th, 1975.

4. Opinion of Consultant in Chest Diseases, November, 1975:

The consultant reviewed the chest x-ray report from the Department of Health which revealed that the left costophrenic angle was obliterated indicating previous pleurisy with residual adhesions. The lung parenchyma showed a slight linear fibrosis. There was no significant disease seen. The pulmonary function studies were within normal limits. Based on that report it was recommended that Mr. Oland's claim not be listed with the Advisory Committee.

-continued-





Claims Review Branch Decision, December, 1975:

The Claims Review Branch denied entitlement in Mr. Oland's claim because there was no evidence of silicosis or any other compensable chest condition.

5. Report of Specialist in Internal Medicine, February, 1977:

(5) The physician advised that Mr. Oland was seen with respect to evaluation of his lungs for asbestosis. The lung scan revealed only borderline changes. The respiratory function tests showed moderate obstructive airway disease. There were some plural changes which were not extensive. A stress test was performed but Mr. Oland was unable to exercise well due to arthritis. Therefore the test was not conclusive. It appeared that Mr. Oland was suffering from a moderate degree of bronchitis and lung damage. Exposure to asbestos may well have contributed to the development of his condition. However the changes could not be conclusively attributed to exposure to asbestos. Should the question of compensation arise the physician felt that Mr. Oland could be considered for a small pension.

6. Report of Advisory Committee on Occupational Chest Disease, August, 1977:

(6) Mr. Oland was examined on August 8th, 1977. It was ascertained that Mr. Oland had attended school with some interruptions until the age of 23. He had been in the army from 1939 until 1946 and then returned to school from 1946 to 1949. He had worked as a draftsman for one year and then as a clerk from 1949 to 1950. He had started working for Canadian Johns Manville May 26th, 1950 and had worked as an inspector and production clerk in the Transit Pipe Department. At the present time Mr. Oland had an easy job but was still in some respects exposed to dust. The total dust exposure was shown as 27 years. The previous history revealed that in December of 1970 Mr. Oland had suffered a myocardial infarction and had been hospitalized for six weeks. He had also suffered degenerative arthritis for the past ten years which had increased in severity. The arthritis affected particularly all joints and the entire spine. Mr. Oland was taking very extensive therapy for rheumatoid arthritis including gold injections and medication. Mr. Oland's activities were grossly limited by extensive arthritis. He was mildly short of breath from walking up

6-4-18  
arthritis curtailed  
by shortness of  
breath, not arthritis



hill or after climbing one or two flights of stairs. Mr. Oland was comfortable at rest. He had a moderate cough with sputum in the morning. Mr. Oland had stopped smoking approximately ten years ago but had smoked heavily since his childhood. On examination there was no clubbing of the fingers. The chest was symmetrical. Air entry was satisfactory. Very few crepitations were present in the left base. There was no wheezing or rhonchi. There was no cyanosis. The heart rate was regular and the heart sounds were well heard with no murmur. An electrocardiogram showed evidence of an old inferior infarct. There was no evidence of congestive failure at the present time. The rest of the examination revealed marked degenerative arthritis in the hand, legs, hips and spine. Pulmonary function tests were carried out which revealed a slight obstructive ventilatory defect. Chest x-rays revealed that both diaphragmatic domes were slightly flattened. There was a questionable plural plaque over the surface of the right diaphragm. Both costo-phrenic angles were shallow and there was a slight pleural fibrosis of both lateral chest walls. The lung parenchyma was clear throughout and there was only minimal linear fibrosis in both bases. A diagnosis was made of slight plural fibrosis with marked degenerative rheumatoid arthritis. There was not enough evidence to warrant a diagnosis of pulmonary asbestosis. It was recommended that Mr. Oland's claim not be allowed at the present time but that he be re-examined in two years.

7. Report of Advisory Committee on Occupational Chest Disease, September, 1979:

It was noted that Mr. Oland had been an employee of Canadian Johns Manville since 1950. At the present time Mr. Oland worked in quality control and spent most of his time in the office. His total asbestos exposure had been 29 years. Mr. Oland's problems were pleural and paranchymal asbestosis and active rheumatoid arthritis. Mr. Oland was taking a considerable number of drugs to control the arthritis. His lung function tests at rest showed a slight obstructive ventilatory defect. Examination of the chest revealed fine inspiratory creptions in the left base and to a lesser degree in the right base. Chest x-ray films, showed slight diffuse interstitial fibrosis and a rather prominent bilateral pleural fibrosis and some pleural calcification. The electrocardiogram showed inverted T waves suggesting previous myocardial eschimia. A diagnosis was made of slight pleural

-continued-





and paraneoplasia asbestosis with advanced rheumatoid arthritis. A disability rating of 10% was recommended with an examination in one year's time.

8. Opinion of Chest Disease Consultant, December, 1979:

The Consultant felt that the findings recorded by the Advisory Committee were inkeeping with the suggested disability rating of 10%.

Claims Review Branch Decision, January, 1980:

The Claims Review Branch concluded that the 10% permanent partial disability award granted to Mr. Oland was inkeeping with the degree of disability residual to the exposure to asbestos. The Union's objection to the amount of the permanent partial disability award was denied.

9. Report of Advisory Committee on Occupational Chest Disease, September, 1980:

⑦ { Mr. Oland was examined on August 25th, 1980. The physical examination revealed a very unhappy man obviously suffering from rheumatoid arthritis. There were degenerative changes noted in his hands and he also complained of pain in most of the other joints. In addition to his arthritis he also complained of shortness of breath, night sweats, easy fatigue ability, and a dry cough with occasional white sputum. Mr. Oland had stopped smoking about 13 years ago. His appetite was fair and his weight was stable. He had no abdominal pain. His sleep was disturbed by a constant cough. His chief complaint other than arthritis was shortness of breath and chest tightness. There was no clubbing or cyanosis. The breath sounds were well heard and of good velocity. Medium crackles were present in the left sub-clavicular area and only partially clear on coughing. The rest of the lung was clear. Heart sounds were well heard and there were no murmurs. Electrocardiogram showed inverted T waves compatible with ischemic changes. There was no evidence of congestive heart failure. The rest of the physical examination revealed deformities due to rheumatoid arthritis. The lung function tests at rest showed slight obstructive ventilatory defect. Chest x-ray films were taken and compared with previous films. There were pleural plaques and slight pleural fibrosis noted on both lateral lung fields. The lung

-continued-





parachemia showed a very slight interstitial fibrosis. A diagnosis was made of pleural changes due to asbestos dust inhalation and probably slight asbestosis. A disability rating of 10% was recommended with a re-examination in one year's time.

BENEFITS:

Mr. Oland was granted a 10% permanent partial disability award amounting to \$101.25 for life from November 23rd, 1979 with \$303.75 given in arrears.



DECISIONS:

10. Claims Review Branch Decision, December 16, 1975  
The Claims Review Branch denied entitlement in Mr. Oland's claim because there was no evidence of silicosis or any other compensable chest condition.
11. Claims Review Branch Decision, January 9, 1980  
The Claims Review Branch concluded that the 10% permanent partial disability award granted to Mr. Oland was in keeping with the degree of disability residual to the exposure to asbestos. The Union's objection to the amount of the permanent partial disability award was denied.

*A. Whitney*  
Mrs. A. Whitney\*jab  
December 17th, 1980





WRITTEN SUBMISSION BY:

MRS. ODETTE DODDS

NOTE: Mrs. Dodds has filed a submission of approximately 300 pages in length. At the Chairman's suggestion, Dr. Mustard has been given the full brief since it contains a lot of medical records. The rest of you have been given excerpts only. The original copy is on file in my office, Room 2213, if you wish to refer to it.

LK





Thank

you.

Violetta Davis & Family  
1981.



From the bottom of my heart, thank you children, grandchildren for your affectionate and support during and after our on going tragedy.

A proud Mother

Love, Mom.

---

I wish to thank Charlie Neilson and his family for their support help and friendship during my husband illness and after his death.

Our best wishes for the future

The Dodds, Briden family

---

Also my thank to Stephen Lewis who needs no introduction for his constant concern during the time of my distress.

"I couldn't wish for a better supporter, writer, speaker, and should I say, a friend." Always John, Odette Dodds and Family.

---

I wish to thank Robert W. Stewart; former Executive Union Director and our Union lawyer David Ublansky for their great co-operation.

---

Gratitude to my dear husband's Doctors.

---

I wish to thank Dr. Stewart Smith for helping me to obtain a widow's pension and bringing a certain Canadian Justice!...

---



Also to Minister of Labour; Dr. Robert Elgie and the Honourable Lincoln M. Alexander for their assistance.

---

Many thanks to Ajax and Pickering General Hospital; the Princess Margaret Hospital and lodge; the Linhurst Hospital and the Toronto General Hospital and Westor Winchester for their considerable care for my dear husband.

---

In addition ot this line up, I should express recognition to Dr. A.C. Wallace for his assistance in the matter at hand.

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To my dear friends and neighbours

Thank you to the Meek's family for helping me in everyway they can and a dear close friend, Beatrice Meachic.

---

Many, many thanks to my dear, best, sincere friend Louise Tarish who helped me in many ways.

Sincerely, Odette Dodds and Family





## GROWING OLD IN BITTERNESS

In 1974, John went to a medical adviser highly recommended, we were hoping to get some help.

I can still picture my husband right now, when one day I brought him the sad news: the medical adviser had put on black and white: I cannot find any good evidence of asbestosis or silicosis!...

John shook his head by telling me, this is a nightmare don't tell me I have to be dead to prove my point, and he was right.

I am growing old in bitterness knowing this painful injustice could have been prevented.

I often ask myself how after such a testimony the person can still work, live with his wife and children and go to church or sleep at night?!...

The good people pay for the bad one would my honesty destroy me too? Only time will tell, and I am willing to take that risk, after all I have nothing to lose, I have lost everything:

my very dear husband!...

Odette Dodds



## A HOPELESS CASE

There was a time that John and I thought very highly of the Medical profession.

We always thought, Doctors support and help each other, we always thought Doctors practiced the Hypocritic Oath!...

A tragedy had to happen to find out how wrong we were.

They say, 2 heads are better than one. The situation I was in, was a hopeless case. In my case I required a dozen heads.

Through my own courage and determination, I succeeded, with the help and guidance of my close advisors and friends, and above all with the mental support of my dear Family and various relations, they never let me down.

Even our Grand-children, they were an affectionate help to me.

Children accept us for what we are.

Grown-ups, can't always do that.

Odette Dodds



## A MIRACLE

Thanks to our devoted honest physicians and allow me to say so, myself, my husband was able to live as long as he did, why and how, I'll never know.

Then after he passed on, I was forced to fight to obtain a widow's pension.

However, the biggest problem was having to content with a form of controversy in medical opinion, was this a matter of diagnosing? or for some reason or other a situation where the medical expert does not wish to make a statement.

As I mentioned on previous occasions, making a small name in History, by bringing a certain Canadian Justice and by exposing the reluctance of medical people to commit themselves in making a justified statement thus not sparing the character of thos at fault, namely the

### Asbestos Companies

I am grateful to the: Canadian Chemical Workers Union,  
"at that time." now: Energy & Chemical Workers Union, Local 26.





Please note

The Workmen's Compensation Board emblem  
carries the work: Justice and humanity

I pity any worker who has to deal with the Workmen's  
Compensation board.

We had a sad experience in 1974. We were lucky John and I  
were able to compete with the critics and pressure. We were  
lucky our family was grown-up and it is a close family and still is.  
We were lucky we had no debts and had a few savings. How many  
couples were as lucky as we were?

What actually happened should not have happened in a civilized  
country.

Sick benefits stopped, unemployment insurance discontinued,  
Canada pension plan, no O.H.I.P. coverage, the worse for a sick  
person and many other workers experienced the same.

Please amke sure it does not happen again, you have the power  
to do so, I don't!...

Odette Dodds



Ministry of Health

Each year John had a x-ray at work, the department of health used to bring their own equipment to the plant. They also knew the danger of asbestos, they are as much to blame as the company by leaving us in the dark!...

Odette Dodds



Big Industry comes to Port Union

on Historic Dixon Farm

"In 1946, Johns-Manville decided to build an asbestos plant in Port Union."

I quote from FACTS AND FOCKLORE written by Mr. John Spilsbury.

Canadian Johns-Manville was 30 years old when they opened in Port Union in May 1948.

To Scarborough Council, indeed most of the people of the Township it was good news. Here was big industry coming to a relatively poor Township, with much needed tax dollars, a million doolar payroll annually and employment for 350 people.

To some local residents it was an aggrevation because an Historic farm had given way to a "noisy, smelly factory." Canadian Johns-Manville had its problems initially. Some people complained of the linseed oil smell from the rock wool manufacturing process, so the Company built a new 200' powered concrete smoke stack in 1949 to replace the 50' metal one.

End of quote It should be mentioned that there was no pressure from any level of Government to correct this problem. etc. etc.





WE ALL BECAME HUMAN GUINEA PIGS

Johns-Manville Company knew before building the asbestos plant about the danger of asbestos, the magic mineral with dust that kills, the biggest industrial killer in history.

Our men were dead ducks before becoming Johns-Manville Asbestos workers. As the years went by Johns-Manville infected our men with industrial diseases, there never was any concern for any of us, not even for our children at school, yours!...

More value was put on the dollar, than on human health.

No wonder Johns-Manville Company still laughs all the way to the bank, while getting away with murder for the last 33 years. Yes, those burracudas certainly have something to laugh about. I repeat, Yes those burracudas certainly have something to laugh about.

Us, wives, widows, we certainly have something to cry about, all our men are dead or dying through Johns-Manville negligence.

Let's all remember this, it is not the one who died who suffer any longer but the one who still is on earth.

Odette Dodds



## DEATH IS A PART OF OUR LIFE

We all know, sooner or later we have to die, the worse part of it is: when you know when and how and John and I knew when and how and I won't even wish my worse enemy to experience what we went through, hell on earth, hell on earth!...

Do you know what it is like to count the years, the months, the weeks, the days, the hours and then the last minutes? Then suddenly it is all over. Everything you worked for, for a life time, there is nothing left of it. Your life looks like a very bad earthquake. You are surrounded by debris and graves, you are scarred for the rest of your days and you still can't do a bloody thing about it.

Do you know what Johns-Manville has done to me, to my children, even to our grand-children, to all of us!...

We all are affected physically, mentally and financially.

First they took the health of our workers, the life of our men, our pride, dignity and respect, they destroyed our home, our rights....



I DEMAND JUSTICE,

WE DEMAND JUSTICE

Time has come for Johns-Manville Company to pay for all the crime they have committed.

It is our turn to laugh all the way to the bank. But all the money and all the tea in China, would never be able to bring our men back; at least give us the satisfaction to live in care and comfort, pride and respect, they even took all that away from us.

Money is power, power is everything, I am only a widow with will power, there is nothing like it!!...

I am Odette Dodds, the late John Dodd's widow something to be proud of!...

Rest in peace, we won't forget you.

Amen.

Odette Dodds





John immigrated to Canada in 1951. Due to a lack of money and health wise, I was unable to come with John and our 2 children, Johnny and Beatrice, so I was left alone in Belgium with our 2 small children for a period of 2 years.

I, Johnny and Beatrice immigrated to Canada in 1953, John found a little bungalow on Lake Ontario and we could see Johns-Manville plant from where we lived. We were as happy as kings, a roof over our heads, an old car that we used to call our Rolls-Royce and icing on our cake.

We had three close neighbours and friends then and the men all worked at Johns-Manville.

They are all dead now, including 2 of the wives.

John was so happy when we were finally united. He said to me one day, now I really have something to work for, but I never thought that 25 years later, his daily work, our bread and butter, icing on our cake, turn to a early grave.

Hard to believe? no!.. That's the way it happened, and worse part of it, the long funeral march still carried on.

Our road to success has been under construction for too long, it is about time it ended, I shall carry on, still have plenty courage, patience and determination. We shall succeed!...

Odette Dodds





DA 91627

FORM 29 - FORMULAIRE 29

THE VITAL STATISTICS ACT - LOI SUR LES STATISTIQUES DE L'ÉTAT CIVIL

# Death Certificate

## Certificat de Décès

NAME - NOM

DODDS, JOHN

SEX - SEXE

MALE

MARITAL STATUS  
ÉTAT CIVIL

MARRIED

DATE OF DEATH  
DE DÉCÈS

JULY 27, 1978

AGE - ÂGE

58 YEARS

PLACE OF DEATH - LIEU DE DÉCÈS

AJAX

REGISTRATION NUMBER  
NUMÉRO D'ENREGISTREMENT

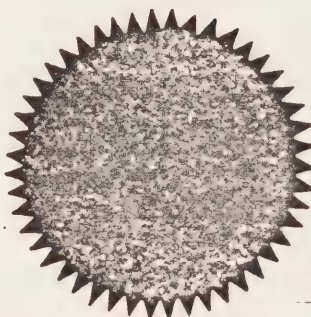
1978-05-035297

DATE OF REGISTRATION  
D'ENREGISTREMENT

AUGUST 1, 1978

ISSUED AT TORONTO, ONTARIO, CANADA  
DELIVRÉ À

AUGUST 15, 1978

*H. A. Vetro*(DEPUTY REGISTRAR GENERAL)  
(ARCHIVISTE GÉNÉRAL ADJOINT)*John J. ...*(REGISTRAR GENERAL)  
(ARCHIVISTE GÉNÉRAL)CERTIFIED EXTRACT FROM DEATH REGISTRY  
EXTRAIT CERTIFIÉ DU REGISTRE DES DÉCÈS*December 12<sup>th</sup> 1979.*

*8 year ago I applied for O.H.I.P. assistance. I received assistance for 1 year till January 1980. I still need assistance, my husband John Dods died July 27<sup>th</sup> 1978, my only income is still Canada pension now, \$124.06 monthly.*

*Thank You.  
Yours truly  
Walter Dods.*

30881544.

*all Merry Xmas and a happy New Year.*





To the.,

Royal commission on matters of health  
and safety arising from the use of asbestos  
in Ontario.

This is a true story of a Johns-Manville  
Scarborough asbestos worker.

Warning.

The true story is sad, depressing, right through

His devotion and honesty to the employer  
destroyed him completely!...

Rest in peace John.....

Witnessed Public.  
Sept. 1981.







# The Late John Dadd's File.

27-1-1920

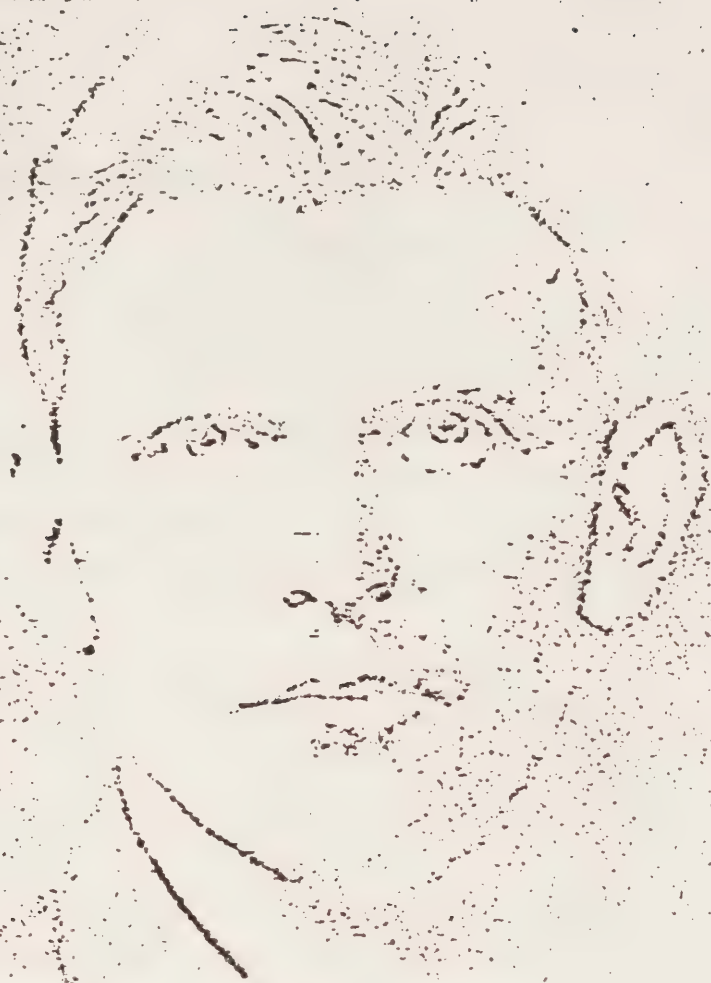
27-7-1870

It was my husband's wish before he died, to put his file into the hands of the right people for the benefit of the workers, public and our country, after his death!...

Please, do make a dead man's wish come true.

Esther Dadd.





John Dodd was a worker at  
the Seabrook plant for 23 years. In 1978,  
he died of asbestosis.

The following account is composed  
of excerpts from the writing of his wife, Odette,  
during the course of his illness.

Odette Dodd.





# Canadian Johns-Manville Co., Limited

Products Division

Toronto Plant  
West Hill P.O.  
Ontario  
(416) 282-1131

November 5, 1974

TO WHOM IT MAY CONCERN:

MR. JOHN DODDS

This is to certify that Mr. John Dodds (Clock No. 1229) has been an employee of the Canadian Johns-Manville Co. Ltd. since December 30, 1952 when he was hired as a Yard Maintenance man.

From Feb. 9, 1953 to March 27, 1961 he was employed in various production capacities.

On March 27, 1961 he transferred to the Maintenance Dep't. as a Mechanics Helper.

On Jan. 22, 1962 he became a Millwright C.

On June 4, 1964 he was promoted to Millwright B.

On March 14, 1966 he was advanced to Millwright A.

On Feb. 10, 1969 he received our then top mechanical rating of Millwright AA.

It should be noted that in the period of 1960 to 1970 there was no Registered Apprenticeship Plan in operation at Johns-Manville's Toronto Plant, and training was done on progressive advancement basis, from Helper through C,B,A, and AA Millwright Groupings.

I am quite prepared to state that, with the exception of the 500 hours related Trade-school to Attendance, Mr. Dodds received between March 27, 1961 and Feb. 10, 1969, considerably more than the present 8000 hours training required by the Apprenticeship Act in equivalent pursuits. During his training and to the present, he has been employed in all phases of mechanical repair and construction millwright work throughout our Plant.

I have known Mr. Dodds through his entire period of employment with Johns-Manville (22 years) and have no hesitation in stating that he is an accomplished Millwright and Industrial Mechanic.

JOHN RIGSBY

Jack started with J-M September 26th, 1951 as a Machinist in E & R. In May 1953 he was promoted to Millwright Group Leader; in April 1961 to Maintenance Supervisor and in December 1972 to Chief Maintenance Supervisor, his current position.

Jack and his wife Betty are residents at 11 Winlaw Place in Markham and are the parents of 4 children - Jim, John, Clyde, a member of our J-M Sales group, and Margaret, and the proud grandparents of 5 grandsons.

Jack has been very active with the Boy Scout movement for several years and more recently has become interested in the Masonic Order.

  
J. W. Rigsby,

Chief Maintenance Supervisor

4.





Illness date - October 7th, 1974.

Co-advised from Company Doctor "Doctor Carson"

lung damage with asbestos, November 22, 1974.

Last day at work, November 27, 1974.

John was forced to retire due to his illnesses at no time was John's intention to quit right on the spot. He was so ill already at the time, he could not carry on his daily work.

The Company nurse was advised right away "Mrs. Audrey Baylis."

In the summary of information from the Workmen's Compensation Board, read on page 3, no. 3 comment about it, also on page 4, no. 6. (Enclosed copies)

Six months later right up to date, Mr. L.B.D. Wilson phoned John to ask him, if he was coming back to work, if not, not only our O.H.I.P. would be cut off in 3 days time but also John would not be considered an active worker and would lose all his benefits and insurance, his life time security will go down the drain. I will carry on for both of us, and I have no regrets.

Odette Dodds



October 7, 1974.

As long as I live I will never forget the day John told me he had asbestosis. I was preparing supper when John came back from work, hugged me and kissed me and asked me to leave what I was doing. "Sit down, he told me. There is something very important I have to tell you. Just listen and don't say anything."

After I heard the verdict, John and I looked at one another and cried for a long time!...



October 22nd, 1974

RE: TODDS, Mr. John  
HF# 247497

Dear Dr.

Thank you for referring this 54 year old man who a couple of weeks ago had a chest xray taken at work and was told that he had evidence of asbestosis and/or silicosis. On direct questioning he says that he has had a cough for some time though he is not sure for how long productive of a small amount of mucoid sputum every day. In addition to this he has been getting more short of breath steadily over the past three or four years. He can walk freely on the flat but gets short of breath climbing two flights of stairs. There is no apyrexial element to this and the dyspnoea is simply related to exertion. At night he may be woken from sleep if he is lying on his back by a choking sensation which is momentary. He is aware, on exertion, of a wheezing noise in his chest. He has not had any previous illnesses of relevance. He had varicose veins removed from both of his legs four years ago and the other three years ago. He has not smoked cigarettes for 12 or 13 years but smokes 3 or 4 cigars a day. He works as a millwright. He is an Englishman who came to Canada in 1951 and before this he worked as a fireman, that is, in a fire brigade. When he came to Canada he went to a construction firm up North and for six months was engaged in drilling coal samples with a diamond drill from rock. He does not know what the firm was looking for. After this he worked in Silverwoods Dairies for a short time and then in a railroad yard and then started to work in 1952 for Johns-Manville. For the first five years he was particularly engaged in poorly asbestos powder and sand and cement out for use and he seems to have been exposed at least to asbestos powder at that time. After five years or so he no longer came in direct contact with asbestos but at least less frequently and is now more engaged in repairing the machinery and construction work working with steel and he handles asbestos now rather seldom.

There is no family history of illness. His bodily functions are normal. His weight has not changed in 10 years. He has not <sup>had</sup> allergies and he takes no drugs. He does take a fair amount of beer and scotch. He has no particular chest pain.

Con't..../2





Con't..../2

RE: DODDS, Mr. John  
HF# 247497

On examination he was a big florid man who was rather overweight. There was no clubbing of the fingers. In the cardiovascular system the pulse was of normal tension and blood pressure was 150/90. The heart did not feel over-active, heart sounds were normal. Peripheral pulses were all present but there is some chronic oedema of both ankles which I think it is due to his venous insufficiency. There were no abnormal lymphglands. The chest moved fairly well. The percussion note was not abnormal and there was no clear evidence of obstructive airways disease except on deep breathing when there was a low pitched rhonchus on both inspiration and expiration on both sides of the chest but moreso on the right. On examination of the neck there is a large goitre which is more prominent on the left side and the trachea is slightly deviated to the right. The abdomen was rather tense and obese but I could feel no definite abnormal masses. He has I think some seborrheic dermatitis, on the front of the chest and probably between the shoulder blades as well though I wasn't quite sure what this was. The throat was clear and the nose was normal.

Radiologically the films are a little hard to interpret because he is such a big solid man. The films taken here really did not clearly suggest any abnormality of the lung fields though the films that you kindly sent along, I admit, are a little suggestive of peripheral lung shadowing. The heart shadow is rather spread out by his high diaphragm and the trachea is clearly deviated to the right by a retrosternal mass. The aorta is somewhat unwound.

I think this man has a significant retrosternal goiter displacing and narrowing the trachea and it may be responsible for some limitation of exercise tolerance as he may not be able to increase his minute ventilation on exercise. I don't think he is thyrotoxic clinically. I do not find any definite evidence of either silicosis or asbestosis and in this particular context I think that asbestos is the one to think about. In particular there are no lymphatic lines and there are no pleural shadows and the pericardial shadow is sharp enough and as I say I am not convinced that there is any pulmonary parenchymal shadowing. I think at any rate this requires investigation. First of all as regards to thyroid I would like to ask my colleague, Dr. ~~W. H. H.~~ to see him and by the time he is seen I hope to have T3 and T4 estimation and a thyroid scan. We will do pulmonary function studies which should include an exercise test and perhaps this can give us some idea as to how much of his exercise is limited by tracheal narrowing. I am also doing a blood picture and electrocardiogram and I would like to reconsider the problem after Dr. ~~W. H. H.~~ has had a chance to see him. My own feeling is that we

Con't.../3



Cont't.... /3

RE: DODDS, Mr. John  
HF# 247497

have no clear evidence of asbestosis here but that the goiter is large enough to merit consideration of treatment.

Thank you again for referring him. I will let you know how things turn out.

Yours sincerely,

/wb  
24-10-74

Dictated but not read.



December 10th, 1974

RE: DODDS, Mr. John  
HF# 247497

Dear Dr.

I enclose a copy of pulmonary function tests of Dr. [REDACTED] examination and of the chest xray report.

As you can see the pulmonary function studies are pretty normal but for a lot of low lung volumes and I do not, myself, think that this is due to asbestosis but simply due to his build and the fact that he is somewhat overweight. If there were asbestosis there would be an xray change and the transfer factor would be low. We have incidentally done a flow volume loop which does not suggest tracheal obstruction.

Meanwhile another symptom has come up which Mr. Dodds had not mentioned to me before though I have asked him specifically. He tells me that he has coughed up some blood intermittently for the past six weeks. He does quite frequently get bleeding from his nose when he blows it and indeed on inspection there is a scarified area in the left naris which may be traumatic. On several occasions however he has coughed up specks of blood and on one occasion four or five weeks ago there was a frank clot. This, of course, is a worrying symptom. I examined him again and I could find nothing any different and looking at his chest xray again there is still no sign of tumor; however I feel myself that any man of this age with coughing up of blood, unless there is a very good reason for it, should be bronchoscoped. I therefore suggested to Mr. Dodds that we do a bronchoscopy and I would be willing to do it without admitting him to hospital but simply getting him up one morning and sending him home afterwards. Mr. Dodds, however, is very reluctant to have this done. I have therefore not insisted but I would suggest to you that this should indeed be contemplated as one cannot rule out of course a carcinoma of the bronchus. Meanwhile, I do not find any evidence of asbestosis or indeed of any industrial lung disease in this man. I think he needs to lose weight. His goitre is being treated but his goitre was not responsible for his shortness of breath and apart from this and his hemoptysis, I do not find any good evidence

Con't.../2

18.





Con't.... /2

RE: DOBBS, Mr. John

REF # 247497

of any disease.

Yours sincerely,

Head, Division of Respiratory Diseases

/wb

12-12-74



November 22, 1974.

For 23 years, John worked for Johns-Manville. He never missed a day or work. When his illness forced him to retire, our Doctor applied for sick benefits. John as refused. Not compensable. John then applied for unemployment insurance. Sorry, not compensable. Six months later a company man phoned to tell John that our O.H.I.P. would be cut off. John said, "You might as well have that too. You took everything else." The company man said, "Why didn't you keep your mouth shut about asbestosis? If you had, there was a way to get financial help." John's answer was, "You want me to kiss your ass. Never! It's about time someone did something."

I shall stick to my guns till the end.



The company man phoned John again, this time to tell him to come to the plant to sign the insurance papers, he was no longer considered to be an active worker, John answer was, no way, it is the place I lost my health, this time it will be your turn to kiss my ass!...

The company man came to our house with the papers. John signed them, this gentleman asked John not be too hard on him, his doing was a company policy, not his own.

He asked John to forgive him and to remember being a company representative, he didn't have the right to have a mind of his own. "I do what I was told to, but I want you to know, you are doing the right thing, if I were in your shoes I would do the same..."

John forgave him and felt sorry for the man, at least John said, I would live in pride and die in dignity!...





We have never been so well off. We have met people in our lives that people dream of.

For years Johns-Manville has been getting away with murders and sweeping the evidence under the carpet. They knew that asbestos caused asbestosis and many other diseases.

Now they have met their master!...

The company Doctor told John there was no cure, no treatment no medication for asbestosis, and, "as for compensation, you might as well forget it. I don't think you will get any."

He said John might as well keep on working. Keep on working, Johns-Manville will kill you a little faster!...

Our only income came from the union - \$30 - \$80 per month.



Ajax, January 1, 1979

at 1:00 a.m.

Unemployment.,

John never failed to pay his premiums for unemployment. In 1974, when he was forced to retire, due to his illness asbestosis, he applied to obtain unemployment, he was not qualified for it, the answer is "honesty" I know so many people who had cheated, abused and never were disqualified to obtain unemployment, I call them dishonest professionals, the same with welfare, not as long as we had 2 hands. We had too much pride and respect for our nre country.



Sunday, January 14th, 1979.

In January 1975, John applied for a disability pension under the Canada pension plan. May 1975, John received a letter about it, and was not eligible to receive a disability benefit under the Canada pension plan.

February 1st, 1978 John applied again and was interviewed in Oshawa about it, it took pretty close to 4 months to get an answer.

John received a cheque at the end of June, 1 year back pay money "\$ 2,179.29." and 1 cheque, \$ 162.03.

In August 1978, a cheque was sent to John \$ 174.18 and it was made the day John died. When I saw it, I cried and I am still crying, when I see it today.

I do suppose a lot of people are in the same boat as we were, if only people would fight for their own rights. It would make our fighting easier and a brighter future for security.





December 27th, 1978.

The illness date was October 7th, 1974, but it was only at the end of November that John was called by the company Doctor to be notified that he had asbestosis. His last day at work was November 27th, 1974.

John was intitled to 25% disability pension and it took pretty close to 8 months before John received his first cheque. Many times John had called the W.C.B. about it, without success first like now.

June 2/75 John findly received a cheque for \$1,270.50, which I have enclosed some copies.

June 9/75 John received a letter from W.C.B. about it.

I was alone to carry on working at: Ajax at Pickering General Hospital. John was very ill at that time, getting worse each day. The only income we had was mine at the Hospital.

Sick benefits were refused to John, unemployment insurance and 6 months later, right up to date (3 days before) Johns-Manville cut our O.H.I.P. "Johns-Manville was unable to get their own way this time, they had to carry out the O.H.I.P. another 3 months." Ajax Hospital helped us as much as they could and carried it out for us.

February 26th, 1976, I quit Ajax Hospital due to health problems and we had to pay privately for our O.H.I.P.

I hope my story will be able to help many.



May 9th, 1978.

Toronto General Hospital

As long as there is life, there is hope, but it is not so, now John has hardly any life into him, there is no more hope, only the hope for me to carry on, and I will.

It is time for us to see and meet one of our writers and for me to make arrangements to the nursing office to have privacy for about 1 1/2 hours.

Everything was perfect. On May 9th, 1978 recording was done on 11th floor in John's room, no one suspected our plans, everything was done just on time.

When the recording was done, John said, what a relief and was proud of it, so am I, and still now.



June 17th, 1978

June 17th, 1978, John came back home from Toronto General Hospital to die at home.

The Lady Doctor made an appointment with me to talk about John's illness, she told me John was a very sick man and was going to get worse. What do you want Mrs. Dodds? I told her that I want to take care of John myself at our home. What will you need to do so? Hospital equipments. That day after I came back home from the Hospital, at 8:00 p.m., I already had some equipments delivered to me.

After I had the talk with the Lady Doctor, I went back to John's room, he was waiting for me with a uncertain look on his face and asked me, what was the verdict? John I said, you are going in a home, our home; and I am going to take care of you we are going to take care of each other till the end. With a smile on his lips, he answered to me, you know that is just what I want. John continued, I had no right anymore to make any decisions now that I am a vegetable. I did not expect you to take care of me, it is so hard for your. Your decision is mine but the one you just made I love you for it, its just what I always wanted.





July 2nd, 1978

John could hardly eat and talk now so I went to a jewelry store and bought a bell that way when I am in the kitchen I could hear his needs. The bell reminds me of a school bell.

I had a new telephone put in his room with push buttons, John was unable to dial the phone anymore and things came so rough that he was only able to use it three times.

I struggled with bars and canes and pulleys and wheelchairs to move him to other positions but even that was useless after, so John said to me with a smile; go in the garage and get 2 - 2x4 and I did, with tears running on my cheeks, I wrapped gib towels around the 2x4 to make sure, not to hurt John. He never did give up, never moan and complained in front of me, and I had to use daily these 2x4.

As long as we were together, I didn't care how hard I had to struggle. I have no regrets, if I had to do it all over again, I would.

Always: John, Odette Dodds & family.



July 14th, 1978

I have spent many hours by John's wheelchair or bedside at home. John stares at me for hours and hours, watching me knit. In two and a half years I have knit 2,500 oz. of wool, to raise money for cancer research or to help people in need. I shall keep using my talent and courage.

I'll never give up.



July 15, 1978

Our life became one long funeral march. Workers are still dying. Everytime John and I hear about another fellow workers death we are dying too. It is a nightmare without end, and all this could be prevented.

They only care about two things - money and production.

Hard to believe? No! That's the way it happened.

For years John tried to put sense into the heads of his fellow workers - to obtain better working conditions, support for workers and their families in case of accident, illness or death; to enjoy early retirement, not an early grave. People still do not realize how serious it is. Through our own experience we are sure that asbestos is a killer. Yes, asbestos is a magic mineral with dust that kills. It is inhaled deep into the tissue of the lungs. The body cannot destroy the particles, but forms scar tissue. The lungs become tough and inelastic. Breathing becomes difficult.

Asbestosis and many other diseases are the results.

We wish we had known 27 years ago what we know now.





July 21, 1978

I had to take John in an ambulance to Ajax, Pickering General Hospital for an x-ray. John became so ill I was unable to bring him back home. He wanted so much to die at home, I told him I would, his answer was, we have both to realize the facts, his illness was out of control now, so I stayed with John morning till night, took care of him most of the time. John continued, I know I am going to have a horrible death, please make sure our 2 kids that I love so much do not see my end.... I would love so much to see and be able to hold my 3 grandsons, before I die. Life is so cruel!... Do you think I deserved this? I have never done any harm to anyone and it is through negligence and greediness I ended up as I am now, a vegetable!...

I told him I am going to keep on fighting. He listens to me with a smile on his face and said you never give up, you never did and never will.

You are so right John, I'll never give up!...

John was right, everything he predicted happened. He suffered like a martyr.

As long as I live I shall never forget it. Here is now over 6 months that John passed away and I am still having nightmares of the end of his death.



July 23, 1978

With tears in his eyes, and so much sadness in his face, John looked at me: "I am sorry dear. You were the one who had to pay for all of this. My last four years were the happiest of my life. You gave me support, care, love, understanding and security. Pretty soon it will be over for me. If only those workers knew hwat I went through.

Pretty soon it will be over for me, but for them, the beginning of an ongoing tragedy."



July 24, 1978

Left home this morning just after 9 a.m. to order a bouquet of 34 pink roses. John's favorite colour. One for each year we have spent together. When he read the card, "From your loving wife, for ever and ever" the tears started to run down his face. All I could say was "Thank you dear."

Today I am remembering all the things we have done to make our life together such a happy one. As long as there is life there is hope.





July 25, 1978

I'll never thought I live to the day to hear my  
husband begging to die!..

September 1981.

Here is now over 3 years John passed away, I never  
had a good night sleep yet.

I will when justice be done!...



July 26, 1978

Much advise was giving to me by my husband before he died: for example, if the W.C.B. tries to put the blame on the thyroid, to remember there are only two men to get in touch with and to trust.



John's 100 years, little calendar pocket booklet

John's prediction, "after his death" 1978. After my death, the workers shall die like flies, many times I shall be in their thoughts and they shall live and work, in fear and shame.

1980. "Starting of a panic."

A very hard year, news is getting around, deaths shall continue.

1985., Things, not getting any better for the war-king class-people.

1990., Getting worse, disaster continues.

2000., This wonderful prosperous new country of ours, going to look like the end of the world. Thanks to Johns'Manville Industrial empire.

I shall rest in peace

Thanks to my bravery, I have nothing to hide. Johns-Manville Company has, asbestos, their killer.





December 19, 1979.

In Memoriam

Dodds. In loving memory of a dear husband, John Dodds, who passed away, July 27, 1978 at Ajax & Pickering General Hospital. "It is sad to walk the road alone, instead of side by side, but to all there comes a moment when the ways of life divide.

You gave me years of happiness, then came sorrow and tears but you left me beautiful memories, I will treasure through the years.

For every loved and missed, wife Odette.

With thanks and appreciation; for all the people who supported and helped us during and after our ongoing tragedy.

Merry Christmas, Best wishes for the coming New Year.

Sincerely -- Odette Dodds and Family.



Monday, January 15, 1979

In July 1975, John received a phone call from the W.C.B., about an appointment for re: rehabilitation. John and Charlie went together July 25th, 1975. They both were very satisfied with the results.

Charlie made a special union meeting about it, inviting John. Both men were stabbed in the back by the workers, they tolde John and Charlie to mind their own business and want to work full time drawing disability pension, it was their business, not theirs, so you see through their own stupidity, good people have to pay for bad ones now.

Everybody could have got the same as John did, till he died, a 100% type of award pension.

Now, the workers are crying they can't get out anymore, out of the plant.

They should think how John Dodds got out of it, determination!...



1975.

# SECURITY FOR YOUR RETIREMENT



...as an Hourly Employee of  
Canadian Johns-Manville Company, Ltd.  
and Subsidiaries

Goody, goody, starting Aug 1<sup>st</sup> 1975  
John going to receive his first retirement cheque  
from Johns-Manville company, all for the amount  
of \$ 66.46. his retirement plan will be \$ 797.52 per  
year, for life?!...

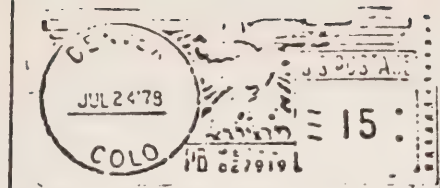
It look like we will be able to continue  
making ends meet!.....

Valerie Dadds.





*Indexed*



*Received - August 3<sup>rd</sup> 1978*  
*"Cheque dated July 31<sup>st</sup> 1978"*

The Johns-Manville pitance monthly retirement cheque



When it's time to retire  
you can look forward to  
an income for life

from your



**RETIREMENT  
PLAN**

*Don't make me laugh!...*

CANADIAN JOHNS MANVILLE		C0H2 0000000193		07-31-78		25583	
DODDS, JOHN		Net		Description		Taxes	
Gross						Year to date	
7646	00	00	7646				
53522	00	00	53522				
Description		Gross	Year to date				
REGULAR		7646	53522				

*John last company monthly retirement cheque. When John died so did the company cheque!...*

*Walter Dodds.*

Retain this statement of your retirement benefits and deductions.



Ajax, December 28th, 1978

September 23rd, 1975. John received a letter from Johns-Manville employee relations manager. "J.B.C. Wilson" to tell John his life insurance policy was reduced. When John died I received a letter with a cheque of \$3,600, 2/3 of John's insurance has been taken off.

I knew that before we made our decision to fight for our own rights and I have no regrets. I preferred to choose John's life that his insurance. I often ask myself, does it really pay to be honest?

I like to know sometimes how many widows did have the life insurance policy cut off?!...

Yesterday December 27th, 1978, there is already 5 months John passed away and no widow's pension yet. I don't mind to have to use our savings to survive, at least John and I, we earned it not only a hard way but an honest way too not only his insurance was reduced but also his life, he deserves to live, for every after and I am going to make sure he does.

Rest in peace John. I shall keep at your good work which was your desires.

Security and justice for everyone.



**Canadian Johns-Manville Co., Limited**

Products Division

Toronto Plant  
5421 Lawrence Ave. E.  
West Hill Post Office  
Ontario M1E 4S3  
(416) 282-1131  
(416) 264-2525

September 23, 1975.

Mr. John Dodds,  
53 Rideout Crescent,  
AJAX, Ontario. L1S 1P9

Dear John:

Enclosed you will find your 'Final Statement of Retirement Income', along with your reduced life insurance policy. I have also enclosed your baptismal certificate.

If you have any questions concerning this information, please let me know.

Regards,



J. R. D. Wilson,  
Employee Relations Manager.





# THE TRAVELERS INSURANCE COMPANY

A Stock Company

HARTFORD



CONNECTICUT

(Hereinafter called the Company)

## CERTIFICATE OF INSURANCE

**THIS IS TO CERTIFY** that the Company has issued and delivered the group policy specified below insuring certain Employees of the Employer specified below and that the Employee named below, a Retired Employee, as defined herein, is insured as hereinafter set forth.

**Employer** CANADIAN JOHNS-MANVILLE COMPANY, LTD.  
and its subsidiary companies named therein

**Policy Number** G 111880C

**Employee** John Dodds

**Effective Date** September 1, 1975

**Beneficiary**—The Beneficiary designated by the Employee in writing and filed at the office of the Employer where the records of the insurance are maintained.

**Amount of Insurance** three thousand six hundred dollars \$ 3,600.00

Other provisions of the group policy principally affecting the insurance of the Employee are printed on the following pages which are made a part of this Certificate.

This individual Certificate is furnished in accordance with and subject to the terms of said group policy and is merely evidence of insurance provided under said group policy which insurance is effective only if the Employee is eligible for insurance and becomes and continues insured in accordance with the terms, provisions and conditions of said policy.

This Certificate replaces any and all Certificates previously issued for delivery to the Employee under the above specified group policy.

The term "Retired Employee" as used herein shall mean a former Employee retired by the Employer who receives retirement income either from the Employer or as a result of service from the Employer.

THE TRAVELERS INSURANCE COMPANY,

*D. H. Mulaher*  
Vice President

## NOTICE TO EMPLOYEE

No agent has any authority to accept or to waive proof of claim required by the policy, nor to extend the time within which notice or proof of claim must be given.

This Certificate should be kept in a safe place known to you and to your beneficiary.

GP-245016 PRINTED IN U.S.A.  
(GC-3901) (GC-3902—4J, 5F, 6D, 22H)

Page 1



## GROUP LIFE INSURANCE—RETIRED EMPLOYEES ONLY

### DEATH BENEFIT

Upon receipt of due proofs of the death of any Employee, provided such death shall occur while the Employee is insured under this policy in accordance with its terms, provisions and conditions, the Company will pay the amount of group life insurance in force on the life of such Employee, in accordance with the "Amount of Insurance" set forth on Page One hereof. The amount payable under this policy upon the death of an Employee shall be paid in accordance with the provision entitled "Beneficiary and Mode of Settlement".

### CONVERSION PRIVILEGE

In case of the termination of any group life insurance hereunder due to termination of an Employee's employment in the classes eligible for such insurance, such Employee shall be entitled to have issued to him by the Company without further evidence of insurability, and upon application made to the Company within Thirty-one days after such termination of insurance and upon the payment of the premium applicable to the class of risks to which he belongs and to the form and amount of the policy at his then attained age, an individual policy of life insurance without disability or other supplementary benefits, in any One of the forms then customarily issued by the Company, except Term Insurance, in an amount not in excess of the amount of such terminated insurance, less, in the case of an Employee who continues in employment with the Employer, any amount of life insurance for which such Employee may be or may become eligible under any other group policy within Thirty-one days after the date of such termination.

If this policy shall be discontinued or shall be amended so as to terminate the group life insurance of any class of insured Employees, any Employee insured hereunder whose group life insurance is terminated by such discontinuance or amendment and who has been insured for the group life insurance for at least Five years prior to the termination of his insurance shall be entitled to have issued to him by the Company an individual policy of life insurance, upon the same conditions and subject to the same limitations as are provided in the next preceding paragraph, except that the amount of such individual policy shall in no event exceed the lesser of

- (1) the amount of the group life insurance on the life of the Employee under this policy at the date of the termination of such insurance less any amount of life insurance for which he may be or may become eligible under any group policy issued or reinstated by the Company or another insurer within Thirty-one days after the date of such termination, and
- (2) Two Thousand Dollars.

In event of the death of the Employee after termination of his group life insurance hereunder and during the Thirty-one day period allowed hereunder for making application for an individual policy and before any such individual policy shall have become effective, the amount of insurance for which such Employee shall have been entitled to make application shall be payable as a death benefit hereunder.

### BENEFICIARY AND MODE OF SETTLEMENT

Any claim for death under this policy shall be paid to the beneficiary designated by the Employee either in One amount or, upon the written election of the Employee upon forms satisfactory to the Company, filed at the office of the Employer where the records of the Employee's insurance under the policy are maintained, in instalments of a fixed amount until the amount of insurance with interest at the rate of Three and one-half per centum per annum is exhausted. The first instalment shall be payable immediately upon receipt of due proofs of death. If there be no such election by the Employee, the beneficiary at the death of the Employee may elect that claim be paid in instalments as herein provided. No such settlement election shall be allowed which shall provide for payments of less than Twenty-five Dollars a month or which shall provide for payments of less than Five Dollars and Seventy-eight Cents a month for each One Thousand Dollars of the insurance.

The Company reserves the right at any Policy Anniversary to change the rate of interest hereunder, subject to a minimum rate of Two per centum per annum, but only with respect to payments for deaths occurring after the date of change; provided that no decrease in the interest rate shall be made effective prior to January 1, 1977.

In the event of the death of the beneficiary after the death of the Employee and before payment of the amount of the insurance or of all the instalments to which the beneficiary may be entitled the unpaid amount of the insurance shall be paid to the executors or administrators of the beneficiary unless the Employee shall have made written request to the contrary in his beneficiary designation.

Any Employee insured hereunder may designate a new beneficiary at any time by filing with the Employer a written request for such change on forms satisfactory to the Company, but such change shall become effective only upon receipt of such request at the office of the Employer where the records of the Employee's insurance under the





policy are maintained. Upon receipt by the Employer of such request the change shall relate back to and take effect as of the date the Employee signed such request whether or not the Employee is living at the time the Employer receives such request but without prejudice to the Company on account of any payment made by it before such request shall have been received.

Payment of any part of the insurance for which there is no beneficiary designated by the Employee or surviving at the death of the Employee shall be made to the executors or administrators of the Employee, provided that the Company may, at its option but without any obligation so to do, pay such part of the insurance as follows:

To the Employee's wife or husband, if living at the death of the Employee; if not living, to the Employee's surviving children, equally; if none survives, to the Employee's surviving parents, equally; if neither survives, to the Employee's executors or administrators.

Any minor's share, unless otherwise provided in a written election by the Employee, may be paid at a rate not exceeding Fifty Dollars a month to such adult or adults as have, in the Company's opinion, assumed the custody and principal support of such minor, but no such payment shall be made after a legal guardian of the estate of such minor shall have been appointed and the Company shall have received due notice of such appointment.

### ASSIGNMENT

No assignment by any Employee of any group life insurance provided under this policy shall be valid.

### TERMINATION OF INSURANCE

All insurance of any Employee covered under this policy shall terminate automatically at the earliest time specified below:

- (1) Upon discontinuance of the group policy.
- (2) Discontinuance of his status as a Retired Employee (as defined).

The insurance under this policy on a Retired Employee shall automatically cease on the date of such termination of his status as a Retired Employee (as defined), as evidenced to the Company by the Employer, whether by notification or by cessation of premium payment on account of such Retired Employee's insurance.

The period that premium is to be paid by the Employer to continue the insurance shall be determined by the Employer on a basis precluding individual selection.

The amounts of insurance as to any such Employee during the periods mentioned above shall be determined in accordance with the provisions of the group policy as to amounts of insurance and shall be subject to change in accordance therewith.





January 2, 1979

- To Johns-Manville Company, point of view it is illegal for a working man to fight for his own rights. If my late husband would have accepted to work full time and draw his disability pension like most of the workers have been doing and still doing it, after his death, I would have obtained a full death insurance, just thinking about it, makes me sick, no wonder, I am bitter, angry and disgusted.

Can you blame me?...



In March 1977, my husband invested \$4,000 in the credit union; in the understanding that after his death this amount would have doubled, in such a way that it would provide me, the survivor with a adequate income.

This all true, I never received anything. It turned out that only if a man passed on, while being an active worker...

Now when a sick Johns-Manville worker is forced to retire due to industrial disease, "in own word due to Johns-Manville negligence" he is able to get his insurance benefit.

Why couldn't the same have happened to John Dodds, for him to have a little luxury during his dying years?



Ajax, November 27, 1979

Please find enclosed copy of death certificate and stuff it!...

Promptly, after receiving the death certificate would you kindly send my cheque, after all its only been 16 months today, since my husband passed away, remember - promptly now!...

I remain, very truly yours.

Odette Dodds

To all.,

Merry Xmas and best wishes for the coming New Year.

Sincerely,

Odette Dodds





January 10th, 1979

Money Talks

November 2, 1978, I received a letter dated October 20, 1978 from Johns-Manville sales corporation to keep me in mind I have enjoyed supplemental health care plan benefits under my husband's name through the C.J. M. retirement group. Things came so rough for John, became completely paralyzed, the equipment John did require we unable to be rented monthly and in one word, you are on your own.

We spend in a day \$1,284.30 from our own money. We couldn't enjoy C.J.M. retirement group fund any longer. The new equipment we bought never was used. I got in touch with "the Starkman Surgical Supply Ltd., 1243 Bathurst St., Toronto 534-8411." The salesman was upset when he heard about the news, John died after his delivery, and told me he would get in touch with me later, he had to talk to his supervisors first about the situation.

A week later, no phone call, so I called him back. With madness, he told me he didn't have the time to discuss the matter with me, and he would let me know about it later.

Today it's January 10th, 1979 and I am still waiting for that phone call but believe me when he heard that we had the cash for that big sale, he came our house less than 1 hour.

Does not matter where you go we seem to have the same problem, money talks!...

P.S. September 1981 - I am still waiting.....



Ajax, Wednesday, April 39, 1980

Dear Dr.

On behalf of myself, my family, Executive Director, Mr. Robert Stewart, Johns-Manville Union President, Mr. Charlie Neilson, our Lawyer, Mr. Daniel Ublansky, please do accept our thanks and appreciation for your co-operation and support by coming to the appeal of the late John Dodds, April 17, 1980.

The appeal started at 9:10 a.m., finished at 3:05 p.m. Before we left the room, I asked the chairman of the board, how long would I have to wait for a verdict!... He replied to me, "I have no idea, maybe 4, 5, 6 weeks, this is not a case all in black and white." Our Lawyer's answer was, we got it in black and white!...

I shall inform you of our progress. Here is my last attempt to: Provincial Minister of Labour, Dr. Robert Elgie and a few latest Johns-Manville newspaper publication.

Thank you Doctor, yours truly

Odette Dodds



In December 1976, John became worse each day. We had plans to spend Xmas over our daughter and son in law, things came so tough for John we had to cancel our Xmas plans, to spend with our children, children in law, grandchildren. John made me promise not to say anything about coming worse, to our family. We agreed to each other, does not matter how hard it is the one who is left behind will continue the fight for asbestos.

I had to get in touch with Dr. office to ask for some help and John and I went to see him several times.

Early in 1977, John's thyroid became enlarged. On the left side of the neck, a lump the size of a grapefruit appeared, on the front of his neck too and another one, on the right side. John had no hope to his neck. He was in agony.

Dr. was pretty sure John had a chocolate cyst and said to us, if there is blood and fluid when I drained your neck, it is a chocolate cyst. We were at that time at the Medical Centre in his office and noticed he didn't have the proper needle for the drainage, so we went to Ajax Hospital, which is just across the Medical Centre and there, he took 30 cc fluid from John's neck. There was blood and fluid, things still came worse so he made the necessary arrangements to send John to Princess Margaret Hospital but there was no bed so we went to Toronto General Hospital. John got his first radiation treatment right away and John found out there was no chocolate cyst but the connection of the thyroid. We waited over a week to obtain a bed over Princess Margaret Hospital and decided to give John 4 radiation treatments daily, but John found out from one of the technicians, it was 4 on the front and 4 on the back, starting 8 a.m. finishing at 5 p.m.





They decided 10 days for radiation. When the 10 days were finished, they decided another 2 1/2 days and John almost went out of his mind. He dais to me, the Medical profession just made a mistake and gave me too many of them.

He knew this time it was his ending and I promised him before he died, it does not matter how hard the consequences would be, I would carry on till the end.

I shall be a winner, not a failure.

Here are some documents, I hope they will be useful to you and for other people in the future.

If you need anything else, don't hesitate to contact me, I am at your disposal.

With thanks and appreciation.

Yours truly,

Odette Dodds.



1962-1978

In 1962, John went back home in England to see his mother, his twin brother and the rest of his family. John's mother just had lost her belonging in a bad storm. Norm and Rosa Shelton took John to the airport. Mr. and Mrs. Shelton have been close friends since we immigrated to Canada.

In Xmas 1977, John was getting worse, so we decided to make plans to go back to England. May 6, 1978, John said to me, this time it will be the last time, but unfortunately everything went wrong with John's illness and May 1st 1978 we went to visit Dr. Robertson at Toronto General Hospital and he sent us right away to be admitted to the Hospital. John walked to the Hospital and a few days later, after his mole was removed from his back, he was unable to stand on his own feet. It was with sadness that John was told by Dr. Sutton, he will be unable to go to England, his condition was too serious.

In the meantime our son Johnny, had made some arrangements to meet us in England during our visit to the family without us knowing it. It was to be a surprise to all of us in England.

John's family never saw our son Johnny (30 years old in 1978). When John heard about the surprise, he cried like a child holding my hand and said, I only wanted to see them all for the last time!...

We were so pleased that he could go, just for us.

When Johnny came back in Canada the same day, he went to visit his Dad at the hospital. Unfortunately John was not getting better and died July 27th 1978.

Rest in peace John!



Norm Shelton was one of the first Johns-Manville asbestos workers who started in the Scarborough plant in 1948. He was sent over to the State for a special retraining program. Norm quit the plant 8 1/2 years later to work in the fire brigade and he still works there. We are still friends.





August 25, 1978

Industrial Chest Disease Service  
Appointment July 5, 1978  
John phoned July 4, 1978

John was unable to keep this appointment due to his illness, he phoned the Chest disease service office to let them know and make another one later. I received a letter addressed to John a long time after his death, and it was the same with Princess Margaret Hospital.

In August 1978, I phoned the thyroid specialist to have an appointment with him, he accepted. I went with our son Johnny, Charlie Neilson was suppose to come with us but another worker died and Charlie was unable to make it.

Johnny and I, talked to the thyroid specialist for 20 minutes. I promised John before he died I would and took some information about John's illness. John asked me to tell the Doctor about the radiation treatments "tell him, he said to me that the Medical profession made a mistake by giving me so many of them, who knows John continued to tell me, my information might in the future, help another poor sole!...

Princess Margaret Hospital

Interview with the thyroid specialist. Johnny and I  
August 25, 1978

Bill Barton died August 24, 1978

Rest in peace, Bill.... Amen.



## Discipline and Gifts

John received discipline, "as Johns-Manville call for 3 times always went back to work," fighting for his own rights, more than ever for a safe, security future for everyone, but never had any credit for it, now that he is dead the workers realise it, and it is too late.



John was always concerned about the welfare of the workers, illness, death, retirement. I still have his homework here, devoted his entire life to help people. When John got sick, no one ever came to see him, except Charlie Neilson. No get well card, only from a dying worker who still kept in touch with me.





December 19, 1979

The day, I wrote this note about Johns-Manville Xmas bonus, we had the visit of our good faithful friend Charlie, he was sitting in one of John's wheel charis and talking to John, when suddenly they looked at each other, stopped talking and both looked at me.

Charlie said to John, I wonder what she is up to now. John's answer was, "she said, she is going to write a book and she cannot even cross the road on her own and of course John mentioned it again, the go train, so they asked me to read it, not only did I surprise them, they also had a good laugh.

John was laughing so much, tears were running down his cheeks, I could still hear him, now!...



1974

You are the one, the one!....

1974 - Christmas bonus - all of \$9.00

In 1974, Christmas, Johns-Manville Co. sent John a cheque for \$9.00. This was called their Christmas bonus.

John went to Dominion and bought a small duck (3 lbs. 2 oz.), 1, 1 lb. butter and 2 lbs. of shortening with the certificate. It was the first cheque and the last one too, that John received.

When I saw the goods my first reaction was to take the goods over to Johns-Manville and stuff those a .....  
like we stuff our christmas turkey and I wish I had.

God bless us all, we need it!...

Amen.



The only sympathy card I received from Johns-Manville workers, except for Charlie Neilson and his family.

Poor soul, he could hardly hold a pen to sign his name.





December 14, 1979. A sick Johns-Manville worker came to visit me and brought me his \$15.00 Christmas turkey certificate for my Xmas.

He was also the only sick Johns-Manville worker who sent me a sympathy card when John passed away. The worker sat in John's blue brocade chair in the living room and cry told me "if you need anything, to be able to carry on, your fight, I'll share with you what I have" and it is with tears running down my cheeks, I thanked him.

December 28, 1979, the worker came back and brought me this time a king size raisin curdbean bread, for my New Year.

December 26, 1979, Charlie and Marie Neilson came to visit me and brought me a gift, stayed a little while with me. They are the only people who kept in touch with me all the time, since John passed away.

You always find who your friends are, when you are in need.



March 1980

I have now received my appeal date "April 17, 1980.  
If my appeal is denied again, I shall reappel it till I do  
obtain a pension!..

I decided once more to not take any change. Would I be  
wise again?

I could hardly wait to find out?



Monday, December 31, 1979

I was disappointed when I heard this appeal December 13, 1979 has been postponed again.

Dr. was on holiday till December 17, 1979 but by the same token, may be it was for the best for us all. In the meantime, I was able to get the pathology's report of first biopsy of John's tumour of the thyroid, let's hope this time, it will be the last evidence we need to complete our case.

I never done so much writing in my life since John passed away and I am willing to do more, if necessary.

P.S. I hope you all have a Merry Xmas and the best for 1980.





Ajax, January 3, 1979

Autopsy Report

As you could see, John was not the type of man to let anyone walk on his feet, taking away his own rights. A life time dream. We keep on fighting this all situation and we still have so much fighting to do about it.

For me, there is no more future anymore but if we keep on fighting, there will be a brighter one for other people, security and justice.

I do not know, how many times John thanked me before he did, at lest he said, I still have a wife, I know she will carry on, my dream.

This Xmas, was a very sad one for all of us and we made the best out of it. I spent if with all my family and family in law.

From Johns-Manville Company, I received 1 Xmas card, it was from Charlie Neilson and his family and 2 dying workers phoned me to see if I needed financial help, offered me their comfort and support, and wished me a Merry Xmas and Happy New Year.

I have been hurt so much, that it does not hurt any more and I know John's spirit still is with me.



June 23, 1978

His last 3 days, John kep repeating to me his wishes, to make sure his body got into the right hands, to find out more about asbestos, that is the only chance we got to save illnesses and life.

His wish was to be cremated and his last words were, "don't forget to pick me up, I want to come back home for ever and ever."

On August 1978, I went to Whitby to pick up John's remains with our children, son and daughter in law, Charlie Neilson and a close friend Jimmy Keenan, a Johns-Manville worker who supported us for many years, the only one left!



Dr. Stopps, Director  
Department of Preventive Medicine,  
and Bio Statistics  
University of Toronto  
Room 305  
50 College Street  
Toronto, Ontario  
M5S 1A8

Dear Dr. Stopps:

I am writing to give you consent to investigate the cause of my husband's death, and to authorize the obtaining of all medical information you may require in order to complete such investigation.

I wish to inform you that my husband was treated at Ajax and Pickering General Hospital (July 21, 1978) and his attending physicians were:

My husband passed away at Ajax and Pickering General Hospital July 27, 1978 at 4:10 a.m. at the age of 58 and worked at Johns-Manville for 23 years. If you require further information you may contact me - Mrs. Odette Dodds, 53 Rideout St. Ajax, Ont. L1S 1P9 and my telephone number is \_\_\_\_\_. I wish to thank you for your interest in this matter and sincerely trust that your investigation will determine the proper cause of death.

P.S. Please be advised of our new phone number and I would appreciate that you would keep it confidential. Thank you.





September 5, 1978. I wrote to Dr. Stopps. I was giving him my consent to investigate my husband's death. I had hoped maybe someone would be able to solve most of our health problems and deaths, but unfortunately when Dr. Stopps received my letters and valuable documents, he phoned me right away and told me, he was not aware, I knew so much about the case and told me, he was sorry but he could not do anything for me, and I should not have any problem to obtain my widow's pension because asbestosis was involved in my husband's death!...

The worse part of it is, you don't trust anyone anymore.

It will take me a little longer alone to solve this painfull injustice but I'll succeed for all of us.



Ajax, Thursday, April 29, 1980

Dear Dr. Elgie:

July 27, 1978, I lost some one very dear to me. My husband, John Dodds died of asbestosis, silicosis, thyroid maglinant Carcinoma, one vocal cord paralysed, spinal cord damage due to radiation treatment, cancer on his back Melanoma and many other problems.

John had a horrible death. Six months later, I received a letter from the Workmens Compensation board, telling me my husband's death was not compensable. The same day, I got in touch with the Union, President Mr. Charlie Neilson, to appeal this 100% injustice.

April 17, 1980, was my appeal date. My only income now is, the Canada pension widow's benefit at \$135.23 monthly.

John immigrated to Canada in 1951. December 30, 1952, John started to work at John's Manville plant in Port Union.

During his entire life in Canada, John never drew, sick benefit, unemployment insurance or welfare! November 27, 1974, John was forced to retire due to his illness, asbestosis.

Sick benefits, unemployment insurance, Canada pension plan, even our O.H.I.P. was cut off.

Through Johns-Manville negligence, not only did he lose his health, but also his life. In 1955-1956 we almost ended in our own grave.

It took my husband and I, many years to find out about asbestos. When we found out about the magic mineral with dust that kills, it was too late. John as a good hard working honest man.



His honesty destroyed him. Before he died, I promised him, I would carry on his good work which was his desires, saving illnesses and life, bringing security, justice and future for every one!

This wonderful country of ours is going down the drain slowly through a Company who never did care about the health and protection of their workers, family and public.

When I lost my husband, I lost everything there is no more future for me!... I have children, grandchildren. I like to see my loved ones, have a future and others too.

It is about time, we realized the facts and changed the law, if we want to keep our country healthy and prosperous. Let's not turn this country to the end of the world. A broken hearted widow.

Yours truly,





Friday night, June 6, 1980

My dear Charlie:

You were right when you said, it is about time I got off my fat ass and write you a thank you letter.

The last time I thanked you was last Xmas 1979, "on a plate." This time it will be only on paper. As you said, I wrote letters to everyone else but you, you are right again.

Thank you on the phone is not enough, a letter is much better, you could read it, over and over again.

On behalf of myself and my family from the bottom on our hearts, we thank you for all you have done for us.

Take care, don't forget.

A very grateful widow, who will never forget your help and support, a-prise to have you as a friend.

Sincerely, yours truly,



Ajax, Monday June 9, 1980

Dear Mr. Alexander:

First I want to congratulate you on your new job.

And, my best wishes and luck. I don't have to tell you that you are going to need it. With your qualifications no doubt about it, you'll be able to solve many of the worker's problem.

Last week, Tuesday, June 3/80, on behalf of myself, Johns-Manville Union President Mr. Charlie Neilson phoned the W.C.B. trying to talk to you about my appeal which was April 17, 1980. The answer was, you were away for a week. After several phone calls to the W.C.B., Mr. Neilson was finally able to get an answer. We are still waiting for Dr. Simpson's report at Princess Margaret Hospital. Two weeks ago, Mr. Neilson phoned, it was the same answer.

Would you please look into this matter. My husband died July 27, 1978 and my only income now is the Canada pension widow's plan, at \$135.23 monthly.

With appreciation,

Yours truly,



Ajax, Friday, June 6, 1980

Dear Dr. Smith:

Over a month ago, I wrote a letter to the Minister of Labour, Dr. Robert Elgie about my late husband's death, which I am sending you a copy. I received no answer to my letter yet, not only for my own concern but also for the concern of the other workers and residents.

The appeal date was, April 17, 1980, but I received also no answer yet.

Johns-Manville Union President Mr. Charlie Neilson made several phone calls to the W.C.B. about it, their answer was they are still waiting for Dr. Simpson's report, Princess Margaret Hospital. It was the same answer over 2 weeks ago. My husband died July 27, 1978.

Could you help me to speed up the W.C.B. decision, for me to obtain a widow's pension?

May 4, 1980, I also met and talked to Mr. Michael Cassidy about my case.

A widow who won't give up!

Yours truly,



Ajax, Wednesday July 23, 1980

Claim - D 10064293 - John Dodds

Dear Dr. Smith:

Thank you for your letter of July 18th, which I received this morning Wednesday, July 23rd.

I am sending you some evidence about my late husband asbestosis and his other health problems. Any other information you need which will help you in the legislation, I am at your disposal.

Thank you Dr. Smith.

Yours truly,





Ajax, Monday, August 11, 1980

Claim - D 10064293 - John Dodds

Dear Dr. Smith:

Just to let you know, I just received your letter this morning, "copy to the Honourable Lincoln Alexander, dated August 7, 1980."

Thank you for your help and interest on my on going tragedy.  
Hope to hear from you soon again.

Yours very truly,



Ajax, Friday night at 11:15 p.m.

August 29, 1980

Dear Dr. Elgie:

Enclosed is a copy for your records from the Workmen's Compensation Board about my husband's death, now compensable.

I don't have to tell you how proud and happy I am, with our victory.

Thank you for your help and concern on my behalf.

With thanks and appreciation.

Yours truly,



December 15, 1980

Workmen's Compensation Board  
2 Bloor Street East  
Toronto, Ontario, M4W 3C3

Attention Mr. L. Alexander

Dear Mr. Alexander:

Thank you for taking a personnel interest in my late husband's case, to bring Canadian justice. I am very grateful to you.

Your appointment as Chairman of the board couldn't have happened at a better time. Now you can realize the problems at the W.C.B. I only wish one thing, which is, that you would have been named Chairman of the Workmen's Compensation Board much earlier.

In closing I would like to wish you and you family a Merry Christmas and best wishes for the New Year.

Yours sincerely,





Ajax, Friday October 3, 1980

at 9:00 p.m.

Canadian Justice

A penny for your thoughts.

Since my husband, John Dodds passed away, July 27, 1978 we have all been working very hard, have had a lot of headaches and pressure.

Through our own determination, we made a dream come true. We brought Canada Justice.

All of us, could be very proud with what we have accomplished.

Each of us have a mixture of qualifications.

Something to be proud of!...

With thanks and appreciation.

Sincerely,



December 15, 1980

Energy and Chemical Workers Union, Local 26  
1620 Dundas St.  
P.O. Box 6246, Station D  
Toronto, Ont. N5B 2Y8

Attention: Mr. Neil Reimer

Dear Mr. Reimer:

Congratulations on your new job. I wish you luck  
in the future.

On December 12, 1980, I attended the 2nd Public Meeting  
in Toronto. I was hoping that I would have the pleasure of  
meeting you there. Possibly at the next one?

Merry Christmas and best wishes for the New Year.

Yours truly,



W.C.B.

Letter dated August 28, 1980

When I received this letter, I was afraid to open it,  
I knew it would be the answer to my appeal!

Just a shock.

Once again - decision non compensable, as continued reading,  
revised decision, then imagine, decided compensable!

John's death was his compensable asbestosis condition.



Ajax, Wednesday, Sept. e, 1980

4:30 p.m.

Claim - D 10064293, John Dodds

Dear Mr. Ranta:

How surprised and pleased was this afternoon, 4:15 p.m., when a Gentleman from the Workmen's Compensation Board knocked on my door to bring me the cheque I was waiting for..

Thank you for your concern. I tried several times to get in touch with you, but the line was busy.

Enclosed is a copy of my late husband's cremation receipt.

Thank you again.

Yours truly,

Odette Dodds and Family





The Workmen's Compensation Board

TORONTO, ONTARIO

DATE 3 SEP 80

N NO. 3204364  
02319

STATEMENT ONLY

WHEN CONTACTING W.C.B. TELEPHONE NO. 416 955-8610  
PLEASE QUOTE - CLAIM NO. 10064293

TYPE OF AWARD PENSION-ODD PAYMENT

TOTAL AMOUNT PAYABLE

9,832.00

PAYABLE TO MRS O DODDS

53 RIDEOUT CRESCENT

**NOT NEGOTIABLE**

The Workmen's Compensation Board

TORONTO, ONTARIO

H DATE 3 SEP 80

N NO. 3204364

THE BANK OF NOVA SCOTIA  
BLOOR AND YONGE

PAY TO THE ORDER OF  
10064293 MRS O DODDS  
53 RIDEOUT CRESCENT  
AJAX ONT



\*\*\*\*\*9,832 DOLLARS & \*\*\*\*\*00 CENTS

AMOUNT \*\*\*\*\*9,832.00

*For O.D.*

⑈3204364⑈ ⑆91132⑈002⑆ 00002⑈13⑈

*September 3rd 1980  
at 4.30 P.M.*

*When the w.c.b. gentleman delivered the cheque \$ 9,832.00, a fortune, I looked at it for many hours. John and I never owned such an amount at one time, he had to be dead for me to have it!..*

*It's a very sad, cruel world....*

*Colette Dodds.*



When I received this widow's pension cheque this month, I was very happy, it couldn't happen at a better month. The 20% increase were spent on my copies, for my brief to the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario.

My pockets are empty once more but I feel like a million dollars with what I have accomplished!...

However, I cannot keep thinking about the other widows. I pray, they too will have a pension.

The Workmen's Compensation Board

TORONTO, ONTARIO

DATE 21 AUG 81

N NO. 4193556  
03596

STATEMENT ONLY

WHEN CONTACTING W.C.B. TELEPHONE NO. 416 965-8804  
PLEASE QUOTE / - CLAIM NO. 10064293  
PENSION PAYMENT DAY NO. 2

TYPE OF AWARD PENSION

TOTAL AMOUNT PAYABLE

492.00

YOU ARE ENTITLED TO A NEW PENSION OF \$492.00. PENSIONS ARE:  
WIDOW(ER) \$492, DEPENDANT CHILD \$136, ORPHAN \$153. PRIOR  
PERIOD ADJUSTMENTS WILL BE MAILED SHORTLY WITH EXPLANATION.

The Workmen's Compensation Board

TORONTO, ONTARIO

G DATE 21 AUG 81

N NO. 4193556

THE BANK OF NOVA SCOTIA  
BLOOR AND YONGE

PAY TO THE ORDER OF  
10064293 DODDS MRS D  
53 RIDEOUT CRESCENT  
AJAX ONT



\*\*\*\*\*492 DOLLARS & \*\*\*\*\*00 CENTS

AMOUNT \*\*\*\*\*492.00\*

*For: [Signature]*  
*[Signature]*

⑈4193556⑈ ⑈91132⑈002⑈00002⑈13⑈

August 26<sup>th</sup> 1981

When I received this widow's pension cheque this month, I was very happy, it couldn't happen at a better month. The 20% increase





Last date at work, November 27, 1974

Originally, we received a false testimony from the official medical adviser.

John got in touch with the dept. of health right away, to arrange an appointment we needed help very badly, John was not getting any better. In the end of January 1975, letter dated Jan. 24, 1975, John received a letter tell him an appointment had been made to be examined February 26, 1975.

Everywhere John went, I was with him. When John finished reading the letter, he said to me, I hope I'll be able to make it till February. He took pretty close of 3 months before John could talk to the head of the department.

1. The head dept. told him he done the right thing, to fight for his own rights.

2. To not let any doctor open him up, he does not stand a chance and 3., John found out that Johns-Manville had known since 1970 his breathing became abnormal, they never said a word about it, and they done the same with all the workers.

Everything John found about his illness through our family doctor and other doctors.

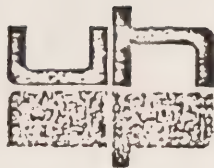
John was always concerned about the benefit of the workers, illness, death or retirement. I still have his home work here, devoted his entire life to help people.

When John got sick, no one ever came to see him, except Charlie Neilson, no get well card, only one, from a dying worker who still keeps in touch with me, no sympathy card after his death only Charlie Neilson and his family, one from a very sick worker now.

In August, I received from Johns-Manville Company a card: donation to Princess Margaret Hospital in John's memoray. By curiosity one day, I am going to get in touch to find out how much, the late John Dodds's life was worth?!...



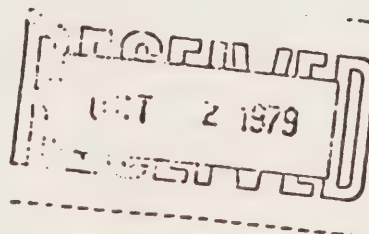




University Hospital  
P.O. Box 5339, Postal Stn., A,  
London, Ontario N6A 5A5  
Owned and Operated by  
London Health Association

339 Windermere  
Phone (519) 673

September 25, 1979.



Mr. Daniel Ublansky,  
Canadian Chemical Workers' Union,  
1620 Dundas Street,  
P.O. Box 6246, Station "D",  
London, Ontario,  
N5B 2Y8

Dear Mr. Ublansky:

I am summarizing herewith my review of the case of Mr. John DODDS. If there is further elaboration on any points that I make below I would be glad to try to amplify them.

In investigating this case, I have contacted individuals at the Ajax and Pickering General Hospital, the Pathologists at the Princess Margaret Hospital and Dr. Alex Ritchie of the Department of Pathology at The University of Toronto who is the pathology consultant for the Workmens' Compensation Board. Dr. Ritchie was kind enough to make all of the material that he had from the Ajax Pickering Hospital together with his own assessment of the case in detail available to me and I spent approximately one-half day at the Banting Institute going over the case from a histological standpoint. I was also able to obtain the x-rays and x-ray reports in addition to all of the information that you submitted to me in June. I will divide my comments into a few major headings but may skip sometimes between them, referring back and forth.

1. The Autopsy Findings:

There is obviously a marked discrepancy between the original autopsy report of Dr. Cass and Dr. Ritchie's subsequent final interpretation of it. I have had access to a more detailed summary of the latter than you submitted to me, although the two differed only in detail.

My general conclusions on the autopsy findings are as follows:

- a) I have no argument with Dr. Ritchie's histological observations on any of the material and should emphasize that Dr. Ritchie is a highly competent individual who did a careful and detailed study on the material in this case.
- b) There is no doubt in my mind that Mr. Dodds died primarily from a very anaplastic carcinoma, apparently arising in thyroid. The report of a second primary malignancy (a melanoma) does not appear to have any bearing on the overall course. I am quite in agreement with other individuals that one cannot make a case for the thyroid carcinoma being in any way related to the asbestosis.

continued....





September 25, 1979.

(2)

Mr. Daniel Ublansky ...

c) It is also obvious, as Dr. Ritchie reported, that the patient also had silicosis, acute bronchopneumonia and pulmonary oedema. He furthermore had fairly severe micronodular cirrhosis of the liver.

d) I think I would be prepared to argue to some extent with Dr. Ritchie on his assessment of the various conditions. Dr. Ritchie declined to comment on the extent of the asbestosis and was careful to say that it was severe in the sections taken. I think I am prepared, on the other hand, to accept the word of Dr. Cass that grossly at autopsy the condition was extensive. I might say parathetically that Dr. Ritchie concluded that the patient probably had fairly extensive acute bronchopneumonia. If he cannot estimate the severity of the asbestosis on the sections he certainly can also not assess the severity of the acute pneumonia. For that matter, we only had a single section of the metastatic thyroid cancer and on that basis would be unable to say how serious this problem was. Again, we are forced to accept the word of the clinicians and the prosector of the autopsy as to the extent of some things.

Another example of where I could disagree at least mildly with Dr. Ritchie was in the assessment of the degree of right heart involvement secondary to the pulmonary disease. Dr. Cass stated that he had a 510 gram heart with a right ventricle that measured 11 mm. in thickness. Dr. Ritchie rejects this latter data as being unlikely+unacceptable. I am, on the other hand, prepared to believe that Dr. Cass measured the thickness of the right ventricle, that he is a competent pathologist, and that the ventricle measured approximately 11 mm. Even if he had been out 50% in a fairly simple measurement, it leaves little doubt that the patient had very severe right ventricular thickening. This points to a severe strain on the right side that may have been related to the silicosis but also to the asbestosis. I do not believe that one can assess the hypertrophy of the ventricle on a microscopic section as suggested by Dr. Ritchie as well as by a measurement of the average ventricular thickening at autopsy.

These points may appear like rather minor quibbles, but they do have a definite bearing on how one interprets the overall picture.

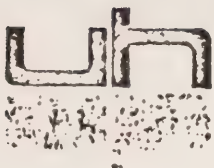
## 2. Overall Assessment of the Severity of the Asbestosis.

The sections available and the report by Dr. Cass leaves very little doubt to me that there was indeed severe asbestosis. There appear to be remarkable discrepancies in the disability as judged in almost every way. The patient was given a 40% disability award and this appears to have been based on functional studies. It seems to me that too much may have been made of the fact that the x-ray findings were never very extensive. I think it is important to realize that in pneumoconiosis in general and in asbestosis in particular the x-ray findings, while they may be important in the original diagnosis, are of no use whatever in estimating the degree of disability. This has been recently emphasized in an article entitled "Summary of Task Force Report on Occupational Respiratory Disease" (pneumoconiosis by G. L. Ostiguy in Canadian Medical Association Journal Vol. 121: No. 4, pgs. 14-421, 1979.

continued...







September 25, 1979.

(3)

Mr. Daniel Ublansky ...

This means essentially that wherever in discussion of this patient's problem the reference is made to the severity of the x-ray findings, one should make it clear that these are irrelevant to the assessment of the case.

3. Unanswered Problems in this Case.

I still am unable to trace down to my own satisfaction some of the most important data that might find useful support to the case you are responsible for. I have been unable to obtain the slides of either the first aspiration biopsy of the patient's thyroid or an alleged second aspirational biopsy. Either one or both of these aspirations was done at the Princess Margaret Hospital. The original tumour diagnosis was papillary carcinoma. This type of tumour is highly suitable for surgical treatment. On the other hand, anaplastic tumour might not be suitable at all. Nonetheless, in a patient who can stand the operation, some reduction of the local tumour burden is beneficial and might have delayed his respiratory trouble.

The key piece of information that I still cannot locate is who made the actual decision not to operate on this patient and why. The Toronto General Hospital's Discharge Form of March 14, 1977, signed by Dr. P. A. McLellan stated that Mr. Dodds was not a surgical candidate. My immediate interpretation of such a statement was that the patient himself was unsuitable to operate on. On the other hand, it may have meant that the tumour was not a suitable one for operation. Until one can contact Dr. McLellan and his colleagues who at the time made this decision I think that we have conflicting stories in our background of information, some saying that he was not operated on because of advanced pulmonary disease and some that he was not operated on because of the type of tumour.

4. Conclusions.

I do not believe that the Chemical Workers' Union should press for compensation on the basis that the patient's asbestosis caused his disease and death directly. I believe that the patient had severe asbestosis that may well have hastened his demise but it may be difficult to prove how much hastening did in fact occur. I believe it would be valuable to find out precisely and reliably why the patient was not operated upon. This problem should be a soluble one. If he was not operated on because of his pulmonary condition, I think it is reasonable to conclude that his subsequent course including radiation damage to his spinal cord and respiratory problems because of the large mass of tumour in his neck may well have played an important contributing role in his death. On this basis, you may find it advisable to press for at least some limited compensation.

I would be glad to go over this report with you and will return all of the material I have when you wish. Dr. Ritchie is a personal friend of mine and I intend to send him a copy of this letter.

Yours sincerely,

*A. C. Wallace*

A. C. Wallace, M.D.

ACW/ed



ENERGY AND CHEMICAL  
WORKERS UNION

R. W. STEWART  
Secretary-Treasurer  
Secrétaire-Trésorier



SYNDICAT DES TRAVAILLEURS  
DE L'ENERGIE ET DE LA CHIMIE

920 Commissioner's Road East  
London, Ontario  
N5Z 3J1  
(519) 685-0310

23rd July, 1980.

Workmen's Compensation Board,  
2 Bloor Street, East,  
Toronto, Ontario.  
M4W 3C3.

ATTENTION: W.A. Molyneux,  
Appeals Administrator

Dear Sirs:

RE: Claim D10064293 - JOHN DODDS

I am in receipt of your letter of June 30, 1980,  
and have had an opportunity to review the material in  
question.

First, I must say that I am most distressed that  
the Appeal Board panel saw fit to distribute the transcript  
of the appeal hearing to Doctors McCracken and Stewart for  
their assessment. The task of assessing the medical evidence  
in this case must fall on the Board. It's not up to either  
Dr. McCracken or Dr. Stewart to evaluate the testimony of  
our two medical witnesses or express opinions on their  
credibility. The Board must examine all the evidence,  
including the sworn testimony of Doctors Khamsi and Syme  
and draw its own conclusions as to the weight to be attached  
to any particular piece of their evidence.

I am even more distressed by the nature and contents  
of the comments made by Doctors Stewart and McCracken in their  
memos directed to the panel. It is not my understanding of  
of the appeal procedures that the full-time medical staff of  
the Board are given the opportunity to present argument in  
support of the decision of the Claims Review Branch subsequent  
to a hearing. I had understood their function to be that of  
providing objective medical opinion, rather than advocate for  
one position or another. Surely that was not the intention  
of the panel in supplying the transcript to Doctors Stewart  
and McCracken. The proper forum for the presentation of  
argument is at a hearing before the Appeal Board in the





WORKMEN'S COMPENSATION BOARD

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presence of all the interested parties not by way of written memos under the guise of medical opinion.

Not satisfied with presenting a rebuttal of the submissions presented on behalf of Mrs. Dodds, both Dr. Stewart and Dr. McCracken, in the course of defending their own opinions, resorted to a vicious attack upon the competence and credibility of the physicians who testified on her behalf. This unseemly attack was completely unprovoked and inflammatory. In reviewing the transcript I found no instance where either witness made any remarks concerning the competence or credibility of any of the medical people who provided reports to the Board in this case.

The panel has provided me with the opportunity to respond to Doctors Stewart and McCracken and, of course, the natural reaction is to respond in kind. However, it would be hypocritical of me to engage in the same sort of unacceptable conduct as those who I have criticized; nevertheless, it is clear now that this whole affair has degenerated into a test of the relative credibility and competence of the medical practitioners on both sides. If that is so, and I see no other possible conclusion that can be drawn, then we must insist that this test be administered on a fair and equitable basis. To date only two physicians have presented themselves before the panel and subjected themselves to examination. Doctors Stewart and McCracken have accused these two physicians of deliberately trying to mislead the panel, of being incompetent, etc., etc. I think the only fair way for the Appeal Board to resolve that issue is to bring all those involved before the panel for examination and cross-examination. I propose that, if the credibility of any physician is to be challenged by this Board or its advisors, then all the doctors who have rendered opinions must subject themselves to similar scrutiny, including Doctors Stewart and McCracken. No one can come to any reasonable conclusion on a matter so sensitive as the credibility of an individual without having the benefit of observing that individual and how he stands up to the rigors of examination. One cannot judge the measure of any individual by reading words on a piece of paper.



WORKMEN'S COMPENSATION BOARD

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Thus in summary, if we are to be afforded a fair and proper opportunity to respond to the memos of Doctors Stewart and McCracken, it simply cannot be done by means of a written reply to the allegations contained therein. That would not solve or settle anything. The competence and credibility of Doctors Khamsi, Syme and all the other medical practitioners who supplied evidence to the Board in support of the claim has been called into question by Doctor Stewart and Doctor McCracken. These medical practitioners must be given a proper opportunity to defend themselves. Similarly, those medical practitioners who have taken the contrary view must subject themselves to the same scrutiny. Then and only then will a sound foundation be laid for the eventual resolution of this matter. It is certainly not my intention to prolong these proceedings unnecessarily and it is not without grave reservations that we take this position on behalf of Mrs. Dodds, but in view of what has transpired since the hearing, we can see no other viable alternative.

If the members of the panel are prepared to accept this suggestion, then I would be happy to discuss the format for such further hearings and any other arrangements that may be necessary. Failing that, I can see no useful purpose in restating the position already taken before the Board at the April hearing or in attempting to furnish the Board with any additional medical opinion in rebuttal to any specific point raised by Dr. Stewart and Dr. McCracken. No doubt that would only lead to further memos and further replies. There must be some finality to these proceedings and some resolution to the issues. I have expressed my view as to how the resolution ought to take place and will await reaction thereto.

Yours very truly,

*Daniel Ublansky*

Daniel Ublansky,  
Legal Counsel.

DU:dh





11C

SUMMARY OF INFORMATION

D10064293, John Dodds

Issue:

The dependents of the late Mr. John Dodds are claiming entitlement to dependency benefits as though his death was related directly or indirectly to his compensable asbestosis.

Diagnosis:

Entitlement: Asbestosis of the lung;

Non-Entitlement: Terminal bronchopneumonia associated with anaplastic carcinoma of the thyroid; malignant melanoma; Biocardial decompensation; Alcoholic hepatitis; Cirrhosis of the liver; Pulmonary oedema.

History:

In 1974 Mr. Dodds, then 54 years of age and employed as a millwright with Canadian Johns Manville Company Limited, West Hill, reported that he had been informed by the company doctor approximately one month previously, that his middle and lower lungs had become infested by the inhalation of asbestos particles. He claimed he had been doing that type of work for twenty-two years. His work history disclosed that he had been doing that type of work for twenty-two years. His work history disclosed that he had been employed in various capacities from December 30, 1952 to November 27, 1974. Early asbestosis was confirmed medically. In May, 1975, the Advisory Committee on Occupational Chest Diseases confirmed slight asbestosis and estimated his disability to be twenty-five per cent. He was re-assessed in April, 1976, and asbestosis and liver enlargement probably due to fatty liver with increased disability was diagnosed. The disability was assessed at forty per cent by the Advisory Committee on Occupational Chest Diseases. The monthly pension payment of twenty-five per cent from October 7, 1974 was increased to forty per cent, and a special supplement of sixty per cent was also approved then.

In January, 1977, his treating internist suggested that he had severe asbestosis of the lungs, and was fully incapacitated. In March, 1977, it was noted that he had anaplastic carcinoma of the thyroid. He was discharged from the Toronto General Hospital on March 14, 1977, with final diagnosis of chronic





alcohol hepatitis, asbestosis of the lung, capillary follicular carcinoma of the thyroid, with anaplastic degeneration. He was then awaiting placement in the Princess Margaret Hospital for intensive radiation therapy. It was medically considered that the diagnosis of carcinoma of the thyroid was in no way related to his asbestosis or to his exposure to asbestos at work. It was medically advised that his non-compensable cancer had rendered him unfit for the labour market, and it was therefore decided to extend the supplement of sixty per cent for a further three months. In July, 1977, his treating internist indicated that he had extensive pulmonary disease which had caused marked incapacitation and had led into cor pulmonale. He had also been diagnosed as having carcinoma of thyroid with severe local infiltration and had done poorly with radiotherapy. He also suffered from cirrhosis of the liver.

Mr. Dodds was hospitalized in the Ajax and Pickering Hospital with respiratory distress and collapse of his left lung. He died on July 27, 1978. The autopsy report gave a provisional anatomical diagnosis of fibrous plaque to diaphragm tumor of thyroid gland, necrotic, (irradiation therapy to same), (malignant melanoma), plural effusion bilateral, pleural effusions left 700 ml., right 500 ml., pulmonary fibrosis, pulmonary oedema, severe, metastatic tumour, right lung, cardiac enlargement 500 grams, right and left ventricles, nodular hypertrophy of prostate. Having examined all of the medical evidence on record, it was the view of the Medical Consultant on Chest Diseases that the late Mr. Dodds had died from complications of thyroid cancer. The pathologist had confirmed that the primary cause of death was obstruction to the necrotic tumor and the radiation in the cervical region, causing obstruction to the deceased's breathing. The pathologist from the University of Toronto thought that it might well be that terminally Mr. Dodds had suffered pulmonary oedema which was in part due to his left ventricular failure in part due to his pneumonia. The late Mr. Dodds' case was considered by the Industrial Chest Disease Service of the Ministry of Labour who concluded that he had died of complications of anaplastic carcinoma of the thyroid, myocardial decompensation and terminal broncho pneumonia. It was also found that his respiratory status as late as May, 1977, would not have precluded any necessary operative procedure. It was concluded that there was no evidence upon which the Board could justify accepting a death claim due directly or indirectly to asbestosis and that decision was confirmed by the Claims Review Branch on January 26, 1979.

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1. Employee's Report of Accident, November, 1974  
He reported that he had been informed by the company doctor approximately one month previously that his middle and lower lungs were infected by inhalation of asbestos particles and the doctor had advised that he file a claim. He had been doing that type of work for twenty-two years. He had never had a similar disability before. At that time he was able to do restricted work.
2. Doctor's Report, Occupational Chest Disease, November, 1974  
He indicated that he had examined the injured employee with shocking, constant cough, and shortness of breath. He had upper respiratory infection. Chest x-rays and sputum examination had been done at the Ajax Hospital and the findings were early asbestosis.
3. Injured Employee's Work History, November, 1974  
It was indicated that the injured employee had been hired on December 30, 1952, and had worked in the yard maintenance as a curer No. 1 machine with asbestos cement and silica. He continued to work with those materials until November 14, 1971, after which he worked with insulating cement. From January 22, 1974 to November 27, 1974, he had again worked with asbestos cement and silica. He was still employed. His total period of service was twenty-one years, ten months, twenty-eight days.
4. Report from Internist, December, 1974  
He enclosed a copy of pulmonary function tests and of the chest x-ray report. He pointed out that the pulmonary function studies were pretty normal, but for a lot of low lung volumes that he himself did not think was due to asbestosis. In view of the fact that the injured employee was coughing up blood he suggested that a bronchoscopy be done. The internist could not find any evidence of asbestosis or indeed of any industrial lung disease. He thought the injured employee needed to lose weight.
5. Doctor's First Report, January, 1975  
He indicated that he had first examined the injured employee on October 7, 1974 after he had received first aid treatment at the Department of Health. Chest x-rays had shown early asbestosis. The injury was sufficient to disable the injured employee from his regular work indefinitely. The injured employee should stay away from exposure to asbestosis.

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6. Letter from Chairman Advisory Committee on Occupational Chest Diseases, May, 1975  
He reported that the findings and recommendations of the Advisory Committee on Occupational Chest Diseases regarding the injured employee were as follows:

He had worked with the accident employer from 1952 to 1974 on transit pipe from 1952 to 1960, and as a millwright from 1960 to 1974. He had had a total exposure of twenty-two years and had retired in November, 1974. The recommendations and diagnosis were asbestosis, slight--disability twenty-five per cent. It was recommended that he be re-examined in one year.

7. Memo from Medical Consultant on Chest Diseases, May, 1975  
He referred to the recommendation of the Advisory Committee on Occupational Chest Diseases and recommended that twenty-five per cent pension be awarded.

Claims Review Branch Decision, October, 1975  
The injured employee's appeal against the twenty-five per cent assessed degree of disability was considered. The Branch confirmed the twenty-five per cent award as representing the injured employee's degree of disability and the supplementary award of 75 per cent. The appeal was denied.

8. Letter from Advisory Committee on Occupational Chest Diseases, April, 1976  
The letter addressed to the Medical Consultant on Chest Diseases disclosed that the findings and recommendations of the Advisory Committee on Occupational Chest Diseases regarding the injured employee were as follows:

1. Asbestosis
2. Liver enlargement, probably due to fatty liver, increased disability, disability 40 per cent.

It was also recommended that the injured employee be re-examined in one year. It was observed that the impression of the examining physician was that the injured employee was drawing 100 per cent compensation at that time, until he was able to locate suitable employment, but it was felt that that situation should not continue indefinitely, and some permanent decision should be made.

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9. Memo from Medical Specialist on Chest Diseases, May, 1976  
He advised that he agreed that the pension should be increased to forty per cent from March 7, 1976 in keeping with the report from the Advisory Committee on Occupational Chest Diseases. He also advised that the special supplement should be adjusted in view of the pension increase. He further advised that the special supplement should be awarded for another six months.
10. Report from Internist, January, 1977  
He reported that the injured employee was then suffering from very severe asbestosis of his lungs. He disclosed that he had been following him for nearly two years, and in his opinion the injured employee was fully incapacitated and unable to return to work. He felt that he deserved full and permanent compensation.
11. Final Summary from Toronto General Hospital, March, 1977  
The report indicated that the 57 year old injured employee presented with three basic problems, namely cirrhosis of the liver dating back to 1943, asbestosis which had been noted approximately two years previously, and a thyroid goitre which was noted to be increased in size approximately two years previously. It was disclosed that a repeat aspiration of the thyroid nodule had been performed. The injured employee had then received one course of radiation therapy at Princess Margaret Hospital and was awaiting placement in the Princess Margaret Hospital for intensive radiation therapy to be given four times daily for nine days. The final diagnoses were chronic alcoholic hepatitis, asbestosis of the lung, papillary follicular carcinoma of the thyroid, with anaplastic degeneration.
12. Progress Notes from Princess Margaret Hospital, March, 1977  
The report indicated that the injured employee with a carcinoma of the thyroid had been admitted for a radiation treatment. He had been known to have a goitre for the previous three years and had been on thyroid replacement, but due to increased nervousness he had discontinued the medication himself. A thyroid scan had been done revealing papillary tumor. In 1974 he had been told that he had asbestosis. The final diagnoses were anaplastic carcinoma of the thyroid, on external radiation therapy, 2. benign hypertension, on diuretics, and 3. asbestosis, 4. possible cirrhosis of the liver.
13. Report from Advisory Committee on Occupational Chest Diseases, June, 1977  
He wrote to the Medical Consultant on Chest Diseases

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explaining that the findings and recommendations of the Advisory Committee in Occupational Chest Diseases regarding the injured employee were as follows:

1. slight asbestosis
2. carcinoma of thyroid gland--treated by radiation therapy at Princess Margaret Hospital and the prognosis was poor.
3. fatty degeneration of the liver.
4. moderate severe restrictive and obstructive ventilatory defect.
5. disability rating from asbestosis at 40 per cent appeared adequate, and  
a recommendation to re-examine in one year.

He further disclosed that recent chest x-ray films were unchanged from previous examination and that there was very little evidence of asbestosis. The injured employee's lung function study showed further deterioration of forced vital capacity indicating moderate to severe restrictive ventilatory defect. His thyroid gland was grossly enlarged, very painful, and from that point of view the injured employee was not suitable for any remunerative employment in the near future. He had constant irritating cough and pain when swallowing. His health was deteriorating and it was not thought that rehabilitation would be feasible.

14. Report from Internist, July, 1977  
He indicated that the injured employee had a multitude of problems, some of which might or might not be associated with his asbestosis. He had extensive pulmonary disease which had caused marked incapacitation, and had led into cor pulmonale. He was diagnosed as having carcinoma of the thyroid with severe local infiltration and had done poorly with radiotherapy. He also suffered from cirrhosis of the liver which might be partly due to excessive ingestion of alcohol, but the internist indicated that he would not be surprised if when the further evidence could be obtained of some relation of cirrhosis with asbestos as a number of his patients had asbestosis also suffered from cirrhosis.
15. Consultation Report from Internist, July, 1978  
The report indicated that the 58 year old injured employee had been admitted to the Ajax and Pickering General Hospital with multiple problems. In summary, he had chronic asbestosis, thyroid malignancy, malignant melanoma and radiation induced paraplegia.
16. Autopsy Report, July, 1978  
The report gave the final diagnosis of fibrous plaque of diaphragm, necrosis of thyroid gland

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and tissues of neck (irradiation therapy to same), metastatic nodule of malignant melanoma, right lung, (solitary), pleural adhesions, severe, bilateral. Pleural effusions, left 700 ml, right 500 ml., pulmonary fibrosis, broncho pneumonia, asbestosis--lungs and bronchial glands, cardiac hypertrophy 510 grams, right and left ventricles, nodular hypertrophy of prostate, chronic urinary cystitis and portal cirrhosis of the liver. It was summarized that the outstanding findings were those of pulmonary asbestosis. The histology of thyroid could not be determined due to irradiation necrosis. A single solitary nodule at the sharp anterior border of the right lung appeared to be of the metastatic melanoma. The final episode was of widespread bronchopneumonia.

17. Report from Pathologist, Ontario Cancer Institute, October, 1978

He reviewed the slides. He indicated that his diagnoses were 1. carcinoma of thyroid gland; A) Extensive local necrosis of tumour in neck, B) Probable solitary metastasis in right lung. 2. Asbestosis. 3. Bronchopneumonia. 4. Cirrhosis, micronodular. 5. Malignant Melanoma--skin of back.

He disclosed that he had not had the opportunity to examine the malignant melanoma which had been removed from the deceased's back in May, 1978, but he thought that a case could be made for the right lung tumour being metastatic from thyroid. He pointed out to the pathologist at the Ajax Pickering Hospital that while his original aspiration biopsy of the goitre done December 16, 1976, showed tumour having a papillary appearance, the Ontario Cancer Institute had an aspiration biopsy of thyroid tumour done on March 22, 1977, which more resembled large cell or giant cell anaplastic carcinoma. He pointed out that many if not all anaplastic thyroid carcinomas appeared to evolve from differentiated papillary or follicular carcinomas. He felt that such evolution may have occurred in the deceased and the histology of the lung metastasis would be quite in keeping with metastatic giant and spindle cell anaplastic thyroid carcinoma.

18. Doctor's Report of Death, November, 1978

He reported that the deceased had died on July 27, 1978 at the Ajax and Pickering General Hospital. His first visit had been in 1974 and the nature of injury was described as severe pulmonary asbestosis, dyspnoeic on exertion. There was steady worsening of his asbestosis, and treatment consisted of supportive measures. The cause of death was given as asbestosis and other morbid conditions were listed as carcinoma of thyroid, and malignant melanoma.

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19. Report from Pathologist, November, 1978  
He submitted copies of the autopsy report on the deceased employee. He noted that at the time that the thyroid tumour was first diagnosed in December, 1976, it was impossible to remove it surgically because of advanced pulmonary disease. There was reasonable opinion that, had surgery been performed at that time, a cure could have resulted.
20. Memo from Medical Specialist in Chest Diseases, December, 1978.  
He disclosed that he had telephoned the pathologist at the Ajax and Pickering General Hospital to inquire about the post-mortem which he had performed on the deceased employee. The pathologist indicated that the deceased had had asbestosis. He also had a thyroid tumour that had been treated with radiation. The pathologist felt that the cause of death in that case was the deceased's difficulty in breathing caused by the excessive reaction to the radiation treatment he had received for his carcinoma of the thyroid. It had caused compression of the trachea leading to his great difficulty in breathing. The pathologist also disclosed that he had forwarded tissue for examination by the pathologist at the Princess Margaret Hospital. That pathologist had also confirmed that the cause of death was in effect a malignancy.
21. Memo from Surgical Consultant, December, 1978  
He traced the history of carcinoma of the thyroid. He also referred to the unsigned "Doctor's Report of Death" which gave the immediate cause of death as asbestosis, and a contributory cause of death as carcinoma of the thyroid. He observed that the pathologist who had carried out the autopsy had stated that the cause of death was post-radiation reaction around the trachea leading to respiratory type of death. He felt that was fully believable. He advised that it would be necessary to secure whatever documentation might be extant concerning the consideration for surgery. He recorded that the type of carcinoma of the thyroid from which the deceased suffered was a very malignant form given to early infiltration of vital structures outside the thyroid. He pointed out that only quite rarely would it be amenable to curative surgery and it was his belief that the majority was managed by palliative radiation.
22. Memo from Medical Specialist in Chest Diseases, December, 1978  
The memo indicated that the Director of the Board's Medical Services had contacted the doctor who had an office at 495 Annette Street. The doctor stated that he was working at the Ajax and Pickering

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General Hospital on duty in the emergency department when the deceased died on July 27, 1978. He had not completed the form. The pathologist at the Ajax and Pickering General Hospital was contacted. He confirmed that the cause of death in his opinion was compression of the trachea by the effects of radiation and a necrotic tumour. That he confirmed was the primary cause of death. A secondary cause of death would be asbestosis. The attending physician felt that the primary cause of death was asbestosis and that the cancer of the thyroid and complications of radiation treatment were only secondary. He said he would complete and sign the report of death.

23. Report from Professor of Pathology, University of Toronto, December, 1978
- He disclosed that he had received from the pathologist of the Ajax and Pickering General Hospital 62 slides stained with haematoxylin and eosin. Portions of lung were present in 16 of the sections and 6 others showed hilar structures. The architecture of the lung was well preserved throughout. He summarized his findings as follows:
1. asbestosis--severe in sections taken, with
    - a) obliterative fibrosis, multifocal, extensive with
      - i) intimal thickening in blood vessels
    - b) centriacinar fibrosis, multifocal, slight
    - c) asbestosis bodies,
      - i) in lung, numerous
      - ii) in hilar nodes, occasional
  2. Pleural plaques--hypocellular, hyaline
  3. Bronchopneumonia--multifocal, acute
  4. Pulmonary congestion, severe.
  5. Pulmonary silicosis--moderate with
    - a) fibrous nodules, multiple, in
      - i) obliterative asbestosis
      - ii) non-fibrotic lung
      - iii) pleural with
        - iv) focal necrosis in occasional nodules
  6. Silicosis of hilar lymph nodes--moderate, with
    - a) fibrous nodules, multiple
  7. Chronic bronchitis - moderate, with
    - a) hyperplasia of mucous glands, moderate
    - b) chronic inflammation, focal, slight
  8. Cirrhosis of liver - micronodular, inactive
  9. Metastatic carcinoma of lung - anaplastic.

He pointed out that though the deceased may have had asbestosis, he could not assess its extent in the lungs, and so could not comment on whether or not his pulmonary function was so far impaired as to make surgery impracticable. The only sections of tumour he had seen were from the lung.

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That tumour was consistent with origin in the thyroid. The slides available to him did not explain the deceased's terminal paraplegia. He knew of no reason to think that either the deceased's carcinoma of the thyroid or his melanoma were related to his exposure to asbestos. The liver showed advanced cirrhosis of a type usually caused by alcohol. The pathologist knew of no evidence that exposure to asbestos increased the risk of developing cirrhosis of the liver. The bronchopneumonia was terminal.

24. Memo from Medical Consultant on Chest Diseases, December, 1978

He referred to the receipt of the pathologist's report as well as the documents relating to the deceased's last admission. He advised that it was then absolutely clear and without any doubt that the cause of death was due to complications consequent upon radiation to the thyroid gland. There was no evidence whatsoever that asbestosis had played a direct or even an indirect role in his final demise. He also pointed out that the medical evidence in the reports from the various doctors and hospitals contained no evidence that radiation treatment to the malignancy was not the treatment of choice initially. The absence of any consultation reports in respect of operable risk in both the Toronto General Hospital notes or the Princess Margaret Hospital notes suggested that that was never a consideration. He pointed out that there was no evidence at all that the deceased had died directly or indirectly from the effects of asbestosis, nor was there sufficient evidence so far that asbestosis prevented surgical interference for thyroid cancer early in 1977.

25. Report from Pathologist, University of Toronto, January, 1979

He indicated that he had no information as to how the deceased had died. He knew from the examination of the slides sent to him by the pathologist taken in conjunction with the autopsy report, and other reports available to him, that at the time of his death the deceased had a very large massive necrotic tumour in his neck, displacing both the trachea and the oesophagus. He had paralysis of at least one vocal chord. He had progressively worsening paralysis more marked in his lower limbs. All those findings were attributable to his carcinoma, or the radiation used so successfully to treat it. In addition he had an acute bronchopneumonia, widespread in the sections available to him. The weights of the lungs recorded in the autopsy report suggested that the pneumonia was bilateral and extensive. In

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In a sense, that pneumonia might well have been the final cause of his death. If there was no question of asbestosis, the pathologist would consider the findings fully adequate to explain his death. He pointed out that certainly the exposure to asbestos did not have anything to do with the development of the deceased's carcinoma of the thyroid or of the complications which resulted from it. He also observed that the deceased had silicosis of his lungs and of his hilar lymph nodes. He could see no reason to assume that the deceased's asbestosis in any way reduced his life expectancy. He suffered from a highly malignant carcinoma difficult to manage. The pathologist explained that he could not determine whether or not the deceased suffered from left ventricular failure. It might well be that terminally he suffered pulmonary oedema which was in part due to his left ventricular failure in part due to his pneumonia.

26. Report from Advisory Committee on Occupational Chest Diseases, January, 1979  
The report disclosed that the Advisory Committee on Occupational Chest Diseases had reviewed in detail the available evidence concerning the diseases and death of the deceased. The Committee concluded that the deceased had not died of asbestosis, but of a terminal bronchopneumonia associated with anaplastic carcinoma of the thyroid, a type of tumour known to be particularly aggressive in nature. The clinical evidence for asbestosis prior to death was circumstantial. It was reminded that the allowance was raised from twenty-five per cent to forty per cent in 1976, because of slight deterioration of pulmonary function tests, but not of such change radiologically. It was also agreed that the deceased could have undergone surgery for thyroid as far as his respiratory system was concerned. The Committee concluded that the deceased had died of complications of anaplastic carcinoma of the thyroid, myocardial decompensation, and terminal bronchopneumonia. His respiratory status as late as May, 1977, would not have precluded any necessary operative procedure.
27. Memo from Director, Medical Branch, January, 1979  
He relayed that the internist had informed him that he had first seen the injured employee in 1974 at which time he had swelling in the neck which he presumed to be a benign goitre. He did not see him again until December 13, 1976, at which time he noted a marked recent growth. Subsequent investigation in January of the thyroid scan showed that there was a cold mass in the neck. The fluid removed

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showed papillary cells which made him consider that he was dealing finally with papillary carcinoma of the thyroid. At that time he made the decision that major thyroid surgery would be hazardous because of the injured employee's asbestosis condition. In March, 1977, the growth became quite rapid and the injured employee was admitted to the Toronto General Hospital. The tumour was huge and the size of the tumour alone would make incubation difficult. The Director of the Medical Branch confirmed the recommendation of the Medical Consultant on Chest Diseases that there was no evidence upon which the acceptance of a death claim due directly or indirectly to asbestosis could be justified.

Claims Review Branch Decision, January, 1979

The dependent's claim to consider the question of whether or not the occupational disease asbestosis was responsible directly or indirectly for the death of the deceased on July 27, 1978, was considered. It was determined that the industrial disease asbestosis for which the deceased was being compensated did not directly or indirectly cause his death. Death benefits were denied.

Benefits:

On May 16, 1975, a 25 per cent pension was awarded. A pension of \$181.50 per month for life was paid from May 7, 1975 with arrears of \$1,270.50.

On May 4, 1976, the pension was increased to 40 per cent. A pension of \$319.50 a month for life was paid from May 7, 1976 with arrears of \$239.50.

Temporary Supplement: On September 25, 1975, a temporary supplement on a 75 per cent basis was awarded for three months. The sum of \$544.50 a month was paid for two months from September 7, 1975 with arrears of \$780.43. That was extended for a further period of six months.

On May 6, 1976, a special supplement of 60 per cent for a period of six months was awarded. The sum of \$435.75 a month was paid from May 7, 1976.

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Decisions:

Claims Review Branch Decision, October, 1975

The injured employee's appeal against the twenty-five per cent assessed degree of disability was considered. The Branch confirmed the twenty-five per cent award as representing the injured employee's degree of disability and the supplementary award of 75 per cent. The appeal was denied.

Claims Review Branch Decision, January, 1979

The dependent's claim to consider the question of whether or not the occupational disease asbestosis was responsible directly or indirectly for the death of the deceased on July 27, 1978, was considered. It was determined that the industrial disease asbestosis for which the deceased was being compensated did not directly or indirectly cause his death. Death benefits were denied.



Inter-Office Communication to William A. Molyneux

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Appeals Administrator

Executive  
From Dr. William J. McCracken, /Director of Medical  
Services Division.

DATE: May 22nd, 1980.

SUBJECT: Response to questions put and transcript  
of appeal case of Mr. John Dodds  
Claim #S10064293

1. Reference will be attached handwritten  
the  
note sent to my attention and / verbal request  
that the transcript be reviewed.
2. Subsequent to this discussion the matter  
was further reviewed with Dr. C. Stewart, Chest  
Disease Consultant and Dr. C. Gray, Specialist in  
Respiraology and part-time Consultant to the Board.
3. A review of the transcript would indicate  
that no new medical evidence has been submitted  
but rather the approach taken by Dr. Syme, by Dr.  
Khamisi and by the legal representative to the  
appeal leads to suggest that the opinions expressed  
by Dr. Syme and Dr. Khamisi be totally accepted  
and the expert opinion supplied by the Chest  
Advisory Committee (a document which is signed  
by members of the Committee in file), the data in  
the file relating to the findings of pulmonary



function studies carried out at various times and the opinions expressed by Dr. C. Stewart all be ignored and considered/<sup>as</sup>invalid and inaccurate. This is, of course, not the case of the matter and this evidence must be weighed against the submission made by the two doctors and the lawyer at the appeal.

4. The fact of the matter is that Mr. Dodds did have radiological evidence of asbestosis and this was reflected by the reports of the Chest Advisory Committee and by the granting of a partial permanent disability pension. At no time was this pension indicative that he was 100% disabled. Supplementary payments were being made primarily on the basis of the socio-economic aspects <sup>of</sup> /the case. In the latter interval of time prior to his death the evidence in file clearly indicates that these additional payments were being made purely on this basis realizing that due to his thyroid disease he would be unable to return to work.

5. At no time was a surgical consultation requested to determine the operability of the thyroid lesion. At no time was a consultation requested by an anaesthetist to determine from the standpoint of anaesthesia whether or not he was





considered as a candidate for anaesthesia for such  
 thyroid surgery. These are areas/beyond the  
 competency of either of the physicians who  
 testified at the hearing.

6. By the time he was admitted to Toronto General Hospital and based upon the clinical findings recorded in the hospital file, it is apparent that the thyroid disease had in all probability progressed to the point where the reason surgical intervention might not have been possible was on the basis of the thyroid lesion having become inoperable rather than on the basis of any decreased pulmonary function and blood gas exchange. It must also be appreciated that this man had other serious disease which would affect his risk from a surgical operative standpoint, these being obesity, hypertension and serious liver disease in the form of cirrohsis. All of these conditions are totally unrelated to his asbestotic condition.

7. In answer to the questions/you have  
 put therefore, the following responses are listed:

7-1 In my opinion the medical decision not to operate on this man from the data in records and from the information obtained from pulmonary



function studies and from the opinions expressed at various times by the Chest Advisory Committee all indicate that his asbestotic condition would not prevent surgery to the thyroid disease rather it is quite evident that at the time of admission to Toronto General Hospital, his thyroid disease had reached the stage where had a surgical consultation and an anaesthetic consultation been requested, there would have been a possibility that the surgeon would have considered the lesion inoperable due to the spread and extent of the thyroid disease.

7-2 In answer to the second question I must therefore conclude that the decision not to operate was not reasonable in that it was not based upon the findings of a surgical and anaesthetic consultation nor was it based on the known pulmonary function studies.

7-3 Relative to the different diagnoses concerning the thyroid cancer, the first diagnosis was made by needle biopsy and the second was made at the time of autopsy. In all probability, both cellular types were present at the time of the needle biopsy and in my opinion this does not indicate any change in the evaluation relative



to the medical and surgical aspects of the thyroid cancer. Finally, as noted, the data in the appeal transcript and a review of the documentation and file has been assessed by the Consultant Chest Diseases, Dr. Stewart, and by the specialist in Respiraology, Dr. C. Gray and their opinions conveyed to my attention.

8. I must conclude, therefore, that from a medical standpoint based upon the data which we have developed and as described in this memo, that in my opinion this man had a degree of asbestosis which could not prevent him from undergoing anaesthesia in the course of treatment of a surgical condition including cancer of the thyroid.

8-1 It would further be my opinion that there would have been a possibility at the time of admission to the Toronto General Hospital that the thyroid cancer had extended to such a degree that should surgical consultation<sup>have</sup>/been obtained the surgeon might well have left it not to approach the problem from a surgical standpoint due to the extension of the thyroid disease.





8-2      This man, therefore, died primarily due to his thyroid cancer disease and the complications which flowed from this disease. He had other serious diseases including obesity, hypertension and cirrohosis of the liver. He had asbestosis, which based upon clinical evaluation by the Chest Advisory Committee and by a number of pulmonary function studies indicated that he had a partial disability due to this condition only.

(Sgd) Dr. McCracken  
M.D., F.R.C.S. (C)





Early 1975, Dr. XX phoned a Union member asking to put more pressure on John Dodds forcing him to return to work. The Union man refused by telling him he was the only man who ever stuck to his guns till the end!...

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On March 3rd, 1977, John received a telephone call from the Workmen's Compensation Board, Dr. XX told him, he was all wrong about the Johns-Manville Company. He would have been better off by going along with the Company and kept on working. John knew he was trying to put words in his mouth, he didn't bite.

John asked him, how sick does a person have to be to draw 100% disability pension, reply: 100%, John repeated the question once again: 100%. After the third, John got the message, death!...

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This last statement by Dr. XX left me shocked but not surprised!...

Rest in Peace John, I won't forget you.



MEMO FROM Dr.C. STEWART

Claim #S10064293 - John Dodds Appeal Board

I have reviewed the entire transcript of 156 pages.

I am assuming from the outset that the Appeal Board will require of this kind of testimony, that statements made, evidence presented and conclusions reached should be factual, consistent with good medical practice and be required to exhibit equal credibility to that routinely demanded by the Board of its own Consultants in the expression of medical opinion.

At one time or another during the day-long testimony, all the Board's consultants in this case which is some degree denegated or otherwise criticized for inaccuracy of conclusions, or for ignoring evidence considered relevant by the claimant and his representatives. Since none of these Consultants were present to challenge these allegations, I am presuming that the Board will expect its Medical Branch to clarify these points and to put them into proper perspective. The only way to do this is to examine the credibility of the two medical witnesses, as well as to establish whether statements made by them coincide with the facts as documented in the medical evidence or with the generally accepted level of medical knowledge in the area discussed. I think this clarification



should proceed prior to analysis of the testimony which almost to the exclusion of all else, emphasizes that he was not a fit surgical candidate because of asbestosis.

DR. SYME:

This witness stated that he first saw the claimant in 1975 and that "he was quite unable to walk", in addition, "the chest condition was begining to lead to heart failure". He attributed all this to asbestosis.

If we consult the original detailed physical examination report by the Advisory Committee of February of 1975, and the report by Dr. Davies of December, 1974, it is very clear that these statements are totally misleading. If we take in consideration the gross obesity of the claimant at the time, it will be seen that the pulmonary function studies were very close to normal and the x-ray only mildly abnormal. His heart shadow was, indeed, enlarged, but he had severe hypertension and a clear history of coronary insufficiency (angina). Also omitted from the testimony was the fact that he had a history of cirrohosis of the liver dating back to 1943. In other words, a mild diminuation of pulmonary function was the least of his problems consequent upon hypertension, coronary insufficiency, cardiomegaly, gross obesity, and alcoholic cirrohosis. This kind of medical evidence,





laced as it is with selective omissions is grossly unfair and victimizes the Appeal Board.

The second item of testimony, page 13, relates to the interpretation of a final summary report from the Toronto General Hospital signed by Dr. P. A. McLellan, included was the statement: "He was admitted on March 9th for consideration of radio-therapy because he is not a surgical candidate". Dr. Syme told the Board that "the usual interpretation of that is that he <sup>was</sup> medically unfit for surgery".

This is blatantly misleading since there is not a single suggestion in this report that he was medically unfit for surgery. He was unfit simply because the cancer had spread beyond the point where it was operable. For example, an individual with cancer of the hilum of the lung who has completely normal pulmonary function is not a surgical candidate simply because it is too late for surgery.

On page 16, Dr. Syme further opines that: "everybody here knows the difficulty in putting a measurement on asbestosis when the person is alive because of the symptoms are quite objective -- in shortness of breath. I don't think there is yet involved a very successful yardstick of measuring it during life". This series of statements is simply false and suggests that the witness has no credible knowledge in the field of pneumoconiosis or pulmonary function testing. First of all, symptoms are



subjective not objective. Dyspnea can be objectively evaluated by measurement of suitable breathing activities in gas diffusion.

I believe the Appeal Board is entitled to fair, impartial and clearly explanatory medical opinion since it is not in a position to challenge medical statements of a general or specific nature, some of which may not be directly related to the case but have significant bearing on it. I believe that the Board should measure the credibility of this witness by the completeness and accuracy of his statements.

DR. KHAMSI:

My concern with this witness is his approach to asbestosis and the knowledge he exhibits. I believe the Board is entitled to closely examine his credibility in his area, since he and the solicitor have both attempted to downgrade the evidence submitted by the Board's Consultants many of whom have had long experience in the field.

First of all there is a letter on file dated July 29, 1977, from Dr. Khamisi which he speculates that cirrohosis may well be associated with asbestos fibre exposure "since a number of his patients with asbestosis also suffer from cirrohosis". This statement will go a long way in establishing the level of Dr. Khamisi's credibility as an informed witness in respect to asbestosis.



Dr. Khamsi goes on to <sup>state</sup> that the cancer of the thyroid could have been cured by surgery and that the necessary surgery was denied solely because of the asbestosis condition, that is on page 46. The decision <sup>based</sup> was made/on "his best clinical judgment".

The Appeal Board should know that it is inconceivable that a physician who has no experience in asbestosis or pneumonconiosis, would deny life-saving surgery to his patient without having obtained the opinion of a competent anaesthetist or respirologist in respect to the risk of the proposed operation. There are very few operations today that cannot be done because of medical risk or medical unsuitability. The operations that present the least hazard are those on tissue outside the abdominal or thoracic cavities. In the attached submission on pre-operative evaluation on pulmonary function, page 306, states in conclusion "the validity of pre-operative evaluation of pulmonary function is based on the assumption that patients at risk can be readily identified." Again, "the patient whose lung function would have been considered to prohibit lung resection in the mid-1950's has been successfully operated on in the 1970's". In another article "Any patient undergoing surgical procedures not involving resection of lung tissue who can maintain gas blood values within normal limits pre-operatively, should be able to maintain these post-operatively if no function in lung tissue is impaired. (pulmonary emboli pneumonitis and atelectasis)" . . .





Again, "Pulmonary function testing should be performed routinely whenever risk is expected. The patient should not be prohibited from necessary surgery; rather post-operative problems should be expected and all efforts made to alleviate them. The patient should not be denied necessary operative procedures on the basis pulmonary function testing placing them in the prohibitive range of the Miller quadrant diagram alone, this term should be changed to 'increased' risk".

#### Summary of the

We reviewed the/Hospital Admissions at the Toronto General Hospital signed by Dr. McLellan and as well the progress notes from the Princess Margaret Hospital signed by Dr. Cane. In neither is there a single allusion to pre-operative evaluation for possible surgery. In addition there are no reports on file of a referral to an anaesthetist or respirologist in December of 1976 when the thyroid was allegedly still benign. We know that less than 12 months previously (March, 1976), there was no diffusion deficit as is usually seen in asbestosis, virtually no obstruction to air flow and the maximum voluntary ventilation (which is a function of overall ventilatory sufficiency) was 71% of predicted for his age and weight. Obviously he was a clear candidate for major surgery with no contra-indication from a point of view of pulmonary function.

When he was next seen by the Committee in March





or May, 1977, the thyroid was grossly enlarged and painful reflecting radiation changes and spread of cancer.

Obviously, under these circumstances, obtaining a reasonable set of pulmonary function studies would be very difficult and increased restriction and obstruction was undoubtedly due to this factor. Even then, however, it was the opinion of the Committee that he could have had whatever necessary operative procedure was necessary.

The credibility of the Advisory Committee has been called into question and the solicitor has used as evidence the findings of severe asbestosis described at autopsy although the Committee found little of this in the chest x-ray. In other words, the sections taken from the lung showed severe asbestosis, but the x-ray and the pulmonary function studies did not support that the rest of the lung was involved in the same way. Dr. Richie made this point on page 62, he stated that "I cannot comment on whether or not his pulmonary function was so far impaired so as to make surgery impractical".

In other words, biopsies do not necessarily reflect the rest of the tissues and it depends on where they were taken from. The lack of a change in the x-ray and the extent of pulmonary impairments suggested that asbestosis in the rest of the lung was not excessive or of mild to moderate degree only.



According to the solicitor the Advisory Committee was mistaken in their assessment effect of the changes in the lung during life. On page 77, he draws our attention to the task force on pneumonconiosis report in relation to disability, assessment, lung function tests, etc. He states that the claimant was rated at 40% during life although the autopsy report shows severe asbestosis. Therefore, he suggested that the Committee's Report should be "viewed with scepticism".

It was further stated that the Committee obviously did not have the benefit of the task force report when they made the assessment in 1977 or 1976. (The report wasn't published until August, 1979.) The Appeal Board should know that this statement is quite untrue since Dr. Cameron Gray Consultant to the Advisory Committee was one of the senior members to the Task Force. In any event the Task Force Report really has no bearing on this case and is really a Red Herring.

It was stated that the findings in autopsy proved that he was 100% disabled from asbestosis at death. The Appeal Board is aware, however, that the impairment of lung function and breathing ability may have little relation to tissue change particularly from isolated biopsies.



PRE-OPERATIVE EVALUATION:

It was stated by the solicitor that Dr. Khamsi was advised by Dr. Simpson of the Toronto General Hospital on March, 1977, that he was not a surgical candidate; however, Dr. Simpson is not a surgeon, he is a radio-therapist, with no qualifications to evaluate surgical risks as in the case of Dr. Khamsi.

The solicitor admits that the critical time for surgery was in December, 1976, not necessarily March, 1977. On page 103, he again says that "The evidence shows that in December, 1976, Mr. Dodds had an operable tumour that was not operated on because of asbestosis."

Once again, there was absolutely no evidence that this was seriously considered by the attending physician and it is inconceivable that a physician in touch with reality would withhold life-saving surgery solely based on a clinical impression of a disease not within his specialty.

On the credibility of Dr. Khamsi, page 107, the solicitor states "I am<sup>not</sup> even certain that the correctness of Dr. Khamsi's decision is really the issue", and further, on page 108 "The issue really is -- when you get down to the core, the issue really isn't who is right or wrong". This statement should be carefully considered in the final analysis of this problem.





When asked if there were any consultation notes with respect to the operable risk available, the solicitor stated "I think again -- all we can do now is to look back. If it doesn't exist, it doesn't exist, but the only explanation that I can offer is that I think it is just not a realistic consideration at that time, probably because of the nature of the tumour at that stage." The only evidence relating to operable risk, according to the solicitor, page 116, "as I say that was - we already saw evidence of that in Dr. Khamisi's letter of December 13, 1976".

This I believe will confirm the fact that no consultations on operable risk were carried out either in the Toronto General or the Princess Margaret Hospitals.

COMPETENCY AND CREDIBILITY:

On page 118, the solicitor says that to question the conclusion that there was insufficient evidence that asbestosis prevented surgical intervention is to question the competency of Dr. Khamisi and Dr. Syme who concluded otherwise. As I emphasized earlier, competency and credibility are vital issues in this case since our own Consultants have been questioned in this regard despite the fact that their opinions have been backed up by objective evidence of one sort or another. What we have established is that there is no objective evidence at all for Dr. Khamisi's assertion that he was unfit due



to asbestosis in December, 1976. On page 144, the solicitor states that "both Dr. Khamsi and Dr. Syme had a much more intimate contact with the patient than the Advisory Committee and much more knowledge of his condition and history."

This is simply false. The Appeal Board will know that the only physicians who carried out credible physical examinations and pulmonary function studies were our Consultants at the Advisory Committee and/or Dr. Davies from Sunnybrook. From these, a reasonable and actual knowledge of his pulmonary impairment and general physical disabilities were drawn.

The solicitor also states that it is somewhat unfair to harp back to the past and rely totally on the evidence for asbestosis prior to death, but the Appeal Board will also know that this is the only way we can estimate pulmonary impairment and operable risk -- The findings at autopsy may have no relation to physiological changes or symptoms during life..

The solicitor went on to say "Now again, I guess this Board is going to have to decide which evidence they think is more reliable on this question. I ask the Board to consider this very carefully." He goes on to say, "but again I suggest to you that if this statement is meant to question the competency of Dr. Khamsi's



qualifications to draw the conclusions that he in fact drew, then I would hope that this Board be very circumspect in making that --- in coming to that conclusion and rejecting Dr. Khamisi's evidence in total".

I would hope that the Appeal Board would not seriously fail to consider the competency of Dr. Khamisi since this is a crucial issue in front of this Board.

The solicitor harps on the fact that allegedly the Advisory Committee and the Board did not consider the pathological findings and I have tried to explain before this is grossly misleading. They have no relation to symptoms or physiological changes in life. The solicitor seems to have no conception of pneumonconiosis and its effects despite of his misuse of the Task Force on pneumonconiosis report. On page 150, he states, "Of course, as far as we are concerned, that memo - - - that report from the Advisory Committee is again of no value."

SUMMARY:

The core of the presentation has been that <sup>the</sup> surgery was not done because of the presence of asbestosis, but even if this decision was right or wrong the fact that it was made in good faith is "all that matters".

We know that no pre-operative risk evaluation





was done and we are asked to believe and to accept in all seriousness that it is an accepted practice to deny a possibly life-saving operation based solely on a "clinical impression by an individual with no first-hand knowledge of the condition allegedly preventing surgery." I stated in a previous memo that I could not conceive of this and the reason why there was never any serious consideration of surgery at any time either in December, 1976 or March, 1977, was simply because of the thyroid condition had progressed beyond surgery.

I have tried to imagine the position of the Board generally as it pertains to advice given on compensable disease. If this is compensable disease exists only in the imagination of the physician giving it. It is difficult not to foresee why lung cancer developing say in a mild silicotic asthmatic, etc., could not be considered inoperable based solely on the arbitrary contention of the attending physician <sup>at</sup> then in his opinion silicosis prevented surgery which might have saved the life of the patient. What this means is we are asked to accept the actions of the attending physician based on good faith alone. In essence, this is what the solicitor in fact asks the Board to do.

#### CONCLUSION:

Not a single item of evidence was presented at





hearing to show that the pulmonary function status of the patient was insufficient to warrant an operation for thyroid cancer. All the evidence shows that the pulmonary function, the gas diffusion, the lung scan done as late as August, 1977, was quite compatible with major anaesthesia.

No right-thinking physician would withhold "life-saving surgery" based on the presence of a pulmonary condition not within his competence to evaluate without requesting a consultation with an anaesthetist or respiratory. The only conclusion to draw is that surgery was never seriously contemplated.

(Sgd) Dr. C. Stewart,

May 20th, 1980.



Newspaper articles by Stephen Lewis.

John said, after my death, you shall find your friends.....

John was right again.

Stephen Lewis came with his secretary Linda Jolly the day John passed away, July 27, 1978.

They stayed with me and our family for over two hours. Charlie Neilson, his family, a very close friend shared our loss with us.

To all of you; Thank you





STEPHEN LEWIS

# Death No. 37 -- and why?

The message at my office was horribly straight-forward: John Dodds, dying in the Ajax-Pickering General Hospital, wanted to see me.

He died just before I got there.

We knew each other only slightly. Our paths had crossed in the fight against industrial disease. He was one of those remarkably brave and decent men, struck down too early in life by circumstances which should never have occurred.

His wife later explained that he was amoning me to urge me never to forget.

John Dodds was 58 years old. His final few months were a nightmare of asbestosis, thyroid cancer and paraplegia.

There was no news story of his passing: No pictures, no eulogies, nothing that would suggest an unusual or special circumstance. But the circumstance was, in fact, part of an ongoing tragedy.

John Dodds is death number 37 among those workers at the Johns-Manville asbestos plant in Scarborough who were exposed to high levels of asbestos contamination in the 1950s, '60s, and early '70s. Like the men before him, he went on compensation and survived not very much longer than that.

Asbestos in 1974. Cancer at the end of 1976. Death in 1978.

Because of the long latency period between original exposure to asbestos dust and the onset of disease, most of the deaths have occurred in the last five years. They come with regularity, sometimes in clusters: There was one last May, one in June, John Dodds in July, and even as I write, there are three men severely ill.

Most of the victims worked for 15 to 25 years at Johns-Manville. John Dodds, for example, arrived in Canada in 1951 and started with J-M in 1952. He left 22 years later, disabled.

## All dead

Mrs. Dodds recollects: "In 1953, I came over with our two children and John found a little bungalow near Lake Ontario. You could see Johns-Manville from our home. We had three close neighbors and friends then, and the men all worked at J-M. They're all dead now."

Charlie Neilson is the president of the chemical workers' union at the plant. For years he's been fighting for greater safety and better compensation. His voice is edgy and brittle when he talks about the frustrations of his job, and I had the sense, with Charlie, that life is one long funeral march:

"I have guys whom the Workmen's Compensation Board has assessed at 25 per cent disability, still working there — even overtime! I've got one man, at 50 per cent disability, who's nearly dying on his feet. . . . No sooner one goes, like John Dodds, then there's another to see. It's a constant job."

Well, let me emphasize that conditions have improved enormously at J-M. There's just no comparison with what once was. Almost every sample now taken of asbestos fibre emissions shows the company to be well within the limits set by government.

Nonetheless, the death of John Dodds triggers, in me a chain of responses and questions.

How do we warn those who have no inkling of what may lie ahead? Johns-Manville used to have an asbestos mining and milling operation near Timmins. It operated for eight years and then was forced to close in 1974-75 because the asbestos contamination was so hazardous.

## 'Tough' limit

The United Asbestos company, with a notorious mill at Matachewan near Kirkland Lake, also shut down.

And are the current occupational health standards for asbestos emissions really safe? Ontario has adopted what is considered a tough limit of two fibres per cubic centimetre. But there are reputable scientists who argue for one-tenth of one fibre as the only acceptable maximum.

Do the companies themselves seem to care? It's hard to believe it.

Look how difficult it was for the asbestos workers at Advocate Mines in Baili Verte, Newfoundland, to win simple concessions on health and safety. Moreover, it is widely rumored in international circles that some of the big asbestos conglomerates are currently negotiating for new mines in the Soviet Union and the Third World where standards aren't so stringent, and enforcement is weak.

Indeed, certain smaller companies have already jumped the gun. One of them, American Asbestos Textiles (Amatex), has, within the last decade — 1969 and 1974 to be exact — opened two new plants just across the Mexican border in Agua Prieta and Ciudad Juarez.

During 1977, two enterprising American journalists discovered that conditions in both plants were deplorable. Ten to 15 years from now, when the workers begin to die, there will be those who speak guardedly of criminal negligence. I'm tired of euphemisms: It's really industrial murder.

Rest in peace, John Dodds. We won't forget you.







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STEPHEN LEWIS

On Friday I wrote a piece for The Star about John Dadds, a 23-year employee with the Johns-Manville company who died last July at the age of 58. At the time of his death, John Dadds was receiving

100-per-cent permanent disability pension from the Workmen's Compensation Board for asbestosis, yet, nearly five months later, his wife still hasn't been granted a widow's pension.

By a sad coincidence, I received, also on Friday, a letter from Helen Barton whose husband, William Barton, was victim number 38 at Johns-Manville. He died last August 24.

At the time of his death, he was receiving a 50-per-cent permanent disability award from the WCB for something called "mixed pneumoconiosis." That simply means that there were many kinds of dust, as well as asbestos fibres, clogging and corroding his lungs.

However, the precise cause of death itself was yet another disease not directly related to occupational exposure, though perhaps related to his existing condition. As a result, Mrs. Barton's application for a widow's pension was turned down.

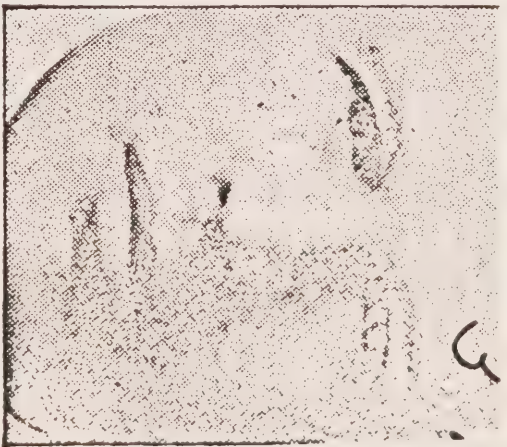
It really makes you wonder. Perhaps the criteria for a widow's pension should be changed in such cases. Perhaps, if a man has worked 25 years in a hazardous environment and develops an occupational illness so severe that he's on a



Mackasey: Temperamental

permanent pension, his widow should not be left penniless if he dies of something else. If the board interpreted "benefit of the doubt" more liberally in the worker's favor, we might not even have to amend the legislation.

Mrs. Barton's last paragraph says it all: "Is this the best that can be done for a hard working man and the family he left behind? For shame."



Macaulay: Untried

And finally, as a special touch of Christmas cheer, I got a letter from Robert Stanfield. No, it wasn't part of the former Tory leader's network. It was really much more intimate than that — Bob wanted a donation from me to help "elect a Progressive Conservative Government."

Gee Bob, I'd really love to, but I'm a little short of cash right now. Have you tried my dad?

# Caught with dope, \$1,000

AJAX (Staff) — A 19-year-old Whitby resident has been fined \$200 in provincial court after police caught him carrying marijuana seeds, a small amount of hash oil and \$1,000 in cash in his gym bag.

David Thompson, 212 Bowman Ave., was arrested Nov. 16 after police found 65.5 grams of seeds, a small set of scales, the hash residue and money.

Judge Edmondson said the facts of the case could have supported the much more

serious charge of possession for the purpose of trafficking. He also noted Thompson has a record of two unrelated offences in 1977.

Thompson said the money was to be used for rent and clothing, as he was moving at the time. The vials, he claimed, were empty.

He was given the alternative sentence of 20 days in jail and placed on probation for a year. A second conviction for marijuana earned Steve Brook, 18, of 51 Rideout St., Ajax, a

\$200 fine and added a conviction to his record. He pleaded guilty to the charge laid Sept. 30 in town.





# Widow won't quit in asbestosis case

Odette Dodds is a woman obsessed. Ever since her husband died at the end of July, 1978, she has spent almost every waking hour pouring over his letters, papers and possessions seeking to assemble a case which will vindicate his life.

It's almost unnerving to listen to her recitation of detail, her consuming need to prove that asbestosis really did contribute to her husband's death after his 23 years as a Johns-Manville employee.

So far she has failed, and has been unable to persuade the Workmen's Compensation Board that she's entitled to a widow's pension. But at this moment in her life, the struggle seems to have much less to do with money than with the principle, forged at death's door between husband and wife, that the death of John Dodds would not be shorn of meaning.

A little paranoid? Perhaps. But look at it this way: A man works all his adult life in a dangerous environment and no one tells him what might happen. He's on substantial compensation when he dies, and then his widow gets nothing.

For Odette Dodds it's as though the world refuses to acknowledge that the workplace killed him.

## No pension

No recognition beyond the grave; not even a pension.

I've written about John Dodds before. He remains one of the most perplexing workmen's compensation cases I've ever come across.

No one involved will dispute that he was receiving compensation for asbestosis for more than three years before his death. Indeed, as far back as Oct. 17, 1975, the board wrote to say the amount of money he was getting was "the equivalent of a pension for total disability."

But when he died, John Dodds also had thyroid cancer, and despite much conflicting medical evidence, the board insisted that it was the cancer, not the asbestosis, which had killed him.

The letter denying Mrs. Dodds' claim for benefits reads in part, "... there was no relationship between the carcinoma of the thyroid and your late husband's asbestosis ... The consensus is that the industrial disease asbestosis for which your husband was being compensated did not directly or indirectly cause his death."

The letter was written on Jan. 23, 1979. From that date to this, Odette Dodds has sustained a personal crusade.

She refuses to rest. She has compiled a beautiful and moving scrapbook of her husband's life: family pictures, love letters, documents, memories of fellow-workers also dead, articles on Johns-Manville, all the shared hopes and shattered dreams of more than 30 years of marriage.

And she's done something else. She's haunted doctors' offices, tracking down, in the process, an entire dossier of new and corroborative medical evidence



STEPHEN LEWIS

which no one has seen in its entirety before.

Some of the material is both depressing and amazing. Poor John Dodds must have felt like a moving laboratory specimen for the last four years of life as he was diagnosed and probed and treated by a dozen different physicians.

But two things have become clearer than ever for me: Everyone acknowledged the severity of the asbestosis, and there were explicit medical links drawn between the asbestosis and the thyroid cancer.

You see, there are a lot of doctors who believe asbestosis was instrumental in John Dodds' death. They have argued that even if the board couldn't accept asbestosis as the direct cause, it was inescapably an indirect cause because the thyroid cancer might have been operable were it not for the asbestosis. Mrs. Dodds should, therefore, receive her pension.

But the board obviously felt that argument was mere conjecture.

Until now. Now Mrs. Dodds — bless her dogged, compulsive tenacity — has uncovered additional evidence.

## Specialist's conclusion

On Dec. 13, 1976, Dr. Firouz Khamisi, the specialist-consultant who first examined John Dodds' thyroid condition, came to the conclusion that whether or not it was benign "... major thyroid surgery would be extremely hazardous to undertake in this man due to very extensive asbestosis ... (He has severe and extensive asbestosis of his lungs and deserves complete compensation.)"

This view was confirmed again a few days before John's death in a consultation report from the Ajax-Pickering General Hospital, involving doctors Syme and Feinstein: "In 1976, he developed a thyroid malignancy which could not be operated on because of his severe lung problems due to asbestosis."

How then can the board maintain the finding that asbestosis "did not directly or indirectly" cause John Dodds' death?

Mrs. Dodds has an appeal scheduled next month when, this time with legal help, she will again try to establish that the benefit of the doubt is on her side.

In the meantime, I sit and read her scrapbook and discover, that in 1959 John Dodds received an award from the company for suggesting an operational improvement which saved Johns-Manville a lot of money.

Perhaps, 20 years later, someone could find some money for his widow.

The Toronto Star  
Sunday Star





There has to be an amendment to the Workmen's Compensation Act. It concerns victims of industrial disease.

I'm not speaking of higher benefits, or a cost-of-living clause, or more generous retroactivity — though, God knows, all would be desirable — but of greater entitlement for the widows and children left behind.

It's a small change really; the costs wouldn't force the Workmen's Compensation Board to pack up shop in bankruptcy. But the change would introduce a sense of fairness and justice which the Act presently excludes.

As things now stand, if a worker is paid compensation equivalent to 100 per cent permanent disability, and then dies, the widow automatically gets full pension, whatever the reason for the death.

Fully compensated

The worker can die from his compensable disease, or from some other disease, or — to carry it to extremes — from falling on the ice or being hit by a truck; but if he was at 100 per cent disability, his widow is fully compensated.

No interminable delays, no expensive appeals, no black nights of tortured anxiety about mortgages and money and the future. The pension doesn't amount to all that much; only \$365 a month. But it's there. Guaranteed.

However, such is not the case if the worker, when he dies, was rated at anything below 100 per cent permanent disability. When that happens, the widow must prove that the cause of death was related to the disease, and, if she can't, she's disqualified. It's as simple as that.

The situation was driven home to me, forcefully, in two recent cases arising from long-term employment at Johns-Manville in Scarborough.

John Dodds died last July 27 after 23½ years at the plant. He was receiving a 40 per cent permanent disability pension for asbestosis, plus an additional 60 per cent granted by the board under a special rehabilitation clause which allows them to award more money "where the impairment of earning capacity . . . is significantly greater than is usual for the nature and degree of his injury."

Was disqualified

John Dodds, therefore, was receiving the maximum he would have received had he been rated at 100 per cent.

Nonetheless, early last week, Mrs. Dodds was disqualified from any widow's pension because the "consensus" of the doctors at the board was that asbestosis, neither directly nor indirectly, had anything to do with the death.

I think they're absolutely wrong, and the decision will be appealed, and as in the case of many such appeals, Mrs. Dodds may have a fighting chance for recognition of her claim.

But that's not really the point of my argument. The point is that surely some pension should be made available to the widow.

There's something inherently screwy



STEPHEN LEWIS

about receiving virtually a full wage in the last years before death in recognition of a crippling compensable disease, and then absolutely nothing for the widow. Not a penny.

And what about Mrs. Helen Barton? Her husband, Bill, died last August 24, age 62. He had 25 years at J-M, and at the time of death, he was on a 50 per cent permanent disability pension for asbestosis.

But again, the board ruled that death was attributable to another disease and Mrs. Barton was denied a widow's pension just a couple of months ago.

So there she was, receiving \$420.53 a month while he was alive, and nothing now that he's gone. It's grossly unfair, and the widows, in such cases, are always bitter and uncomprehending.

And then there's the very recent case of Ed Compton. He passed away a week ago Saturday at age 61. He was a widower with five children of various ages who survive him. Two, at least, were significantly dependent upon their father.

Straightforward

Ed Compton worked at J-M for nearly 30 years. When he died he, too, was on a 50 per cent permanent disability pension for asbestosis. But suppose the WCB finds that asbestosis was not the cause of death, what then? It's not complicated: Nothing.

The amendment required is utterly straightforward.

If a victim of an industrial disease is permanently disabled to any significant degree at the time of death — let us say 25 per cent or greater — then a pension should go, automatically, to the widow and children.

If a full pension is unacceptable to the legislators, let it be pro-rated to reflect, at least, the same percentage as the disability. It won't be much, but it will be something.

For once, I am not making an all-out assault on the Workmen's Compensation Board. This is a matter of legislation. It is a matter for the ministry of labor and the MPP's.

We are not talking about back injuries, and hand injuries, and more traditional on-the-job accidents. We are talking about insidious and devastating occupational diseases which are caused by exposure to chemical contamination and from which, very often, there is no chance of recovery. The situation is so special that there is a whole section of the Workmen's Compensation Act called Industrial Disease.

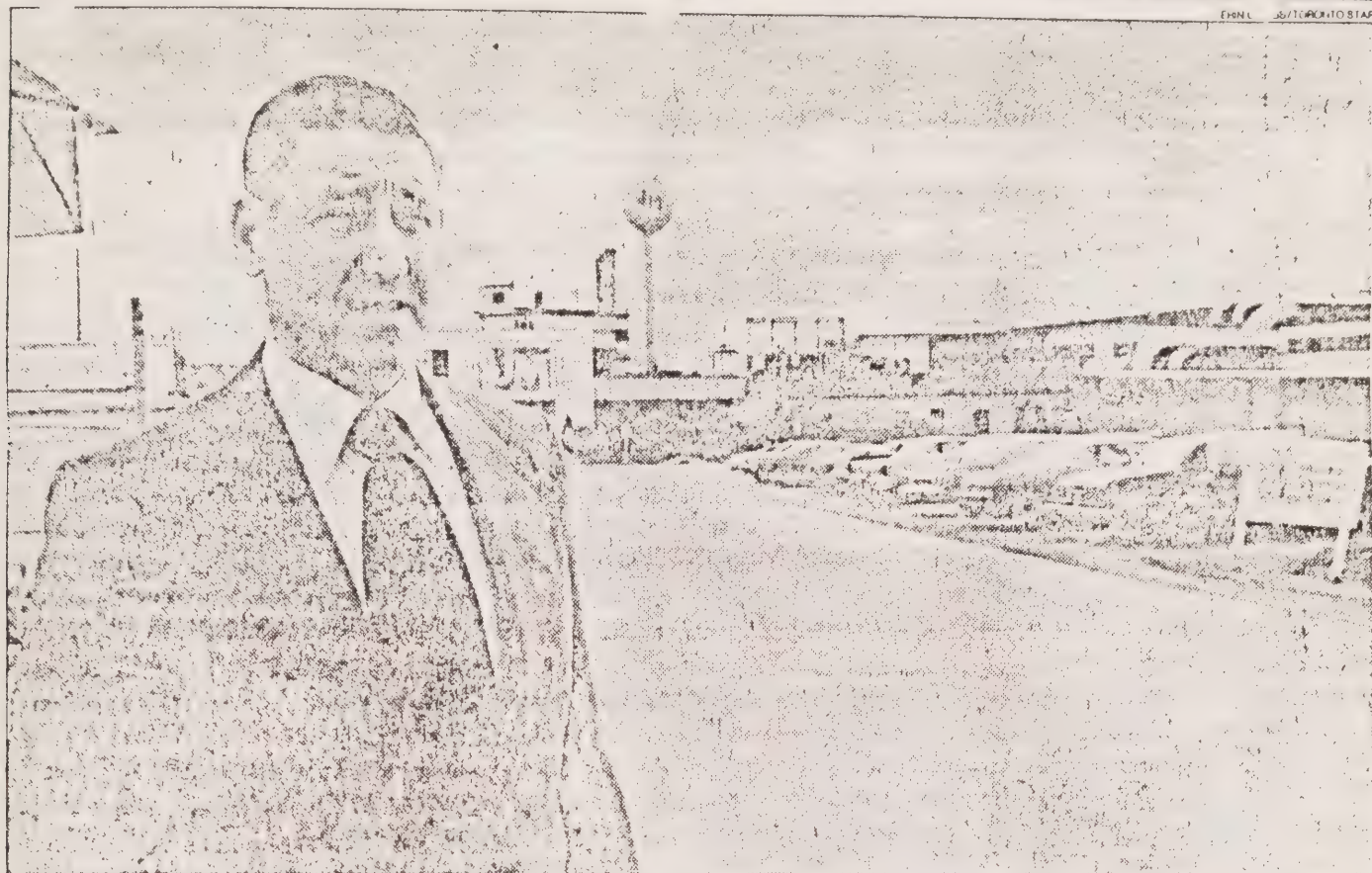
Under the Act, widows and other dependants forfeit their right to sue the company for damages in the event of injury, disease, or death.

Must they also forfeit their right to a modest and life-giving pension?

*Sunday Star, February 4 - 1979*  
*Ed Compton worked at J-M, 30 years. Died January 27, 1979. For nearly 30 years. "Just a couple of months ago."*  
*Bill Barton worked at J-M, 25 years. Died August 24, 1978. For 25 years. "Just a couple of months ago."*  
**WIDOWS NEED IS SIMPLE JUSTICE ON THEIR PENSIONS**







Union president Charlie Neilson in front of Johns-Manville plant in Scarborough

# What does it take to obtain humane response from WCB?

I don't really want to have to write columns like this anymore. It's depressing and repetitive. But so help me, so long as the Workmen's Compensation Board fails to respond, in an urgent and human way, to tragedies in the workplace, I shall keep at it. And so shall many others.

One of them is Charlie Neilson, the rugged and indefatigable president of the union at Johns-Manville in Scarborough. Sometimes I just don't know how he survives from month to month when every knock at the door seems to bring news of one of his old assembly-line chums having succumbed to death or disease.

Charlie phoned me early Tuesday morning. His conversation, as always, was sparse and to the point: "We've got to do something about the John Dodds case; I've even called Michael Starr, but we still haven't got a decision. My God, Stephen, it's nearly five months."

## Died in July

Indeed it is. John Dodds died on July 27, 1978, at the age of 58. He had worked 23½ years for Johns-Manville; worked until 1974 when asbestosis choked his lungs and forced him to retire. In the years immediately before death, the asbestosis was so bad that John Dodds was on a 100-per-cent permanent disability pension from the compensation board.

And now it's mid-December, coming, close to Christmas, and his wife of more than 30 years still hasn't been granted a widow's pension.

Dec nt, eh? And seized with that compassion and speed of response for which the board is renowned.

It's hard not to be bitter on Mrs. Dodds' behalf. John Dodds gave his working life to industry, and in return, industry took the rest of his life from him. I remember visiting their home on the day of the funeral, and Mrs. Dodds



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took me to John's bedroom which looked for all the world like an intensive care unit transported direct from hospital.

He had died a particularly terrible death. Not only was there acute asbestosis, but in the last year of his life there was also cancer of the thyroid and a malignant tumor in the lung. Radiation treatment had left him paralyzed from the waist down, he was incontinent, and his wife struggled with bars, and pulleys and canes and wheelchair to move him, inch by inch, to other positions.

You would think, somehow, that in the case of John Dodds, death number 37 from exposure to asbestos at Johns-Manville, it would be possible for the board to react with dispatch. But you would think wrong.

## Three months later

Oh, it isn't that they didn't know. As always, on the very day a colleague dies, Charlie Neilson phoned the board to tell them. True, they couldn't just take Charlie's word for it; they had to wait for the medical reports, but they got those in September, and it's three months later, and Mrs. Dodds still doesn't have a widow's pension.

Not that her husband's passing wasn't acknowledged. She got a letter from the board at the end of September with "Mrs. Dodds" typed in, and the first paragraph read as follows:

"We were very sorry to learn of the death of your spouse and are making inquiries to determine your entitlement to benefits under the Workmen's Compensation Act."



John Dodds; Widow still waiting

Well, fair enough, I guess. The Board's a pretty big outfit and a lot of people die, and you can't expect them to type a personal letter.

But what is that stuff about "... making inquiries to determine your entitlement."

I have the documents in front of me. The immediate cause of death is listed in the certificate as asbestosis. The nature of the "injury" is listed as "severe pulmonary asbestosis." The symptoms and course of the disease is described as "steady worsening of asbestosis."

## Autopsy report

And if that's not enough, there's the autopsy, with three doctors in attendance. The report reads as though almost every organ of poor John Dodds' body was shot through with asbestos fibres and "severe" asbestosis. The diaphragm,

both lungs, the entire respiratory system, the bronchi, until finally, under the heading of "summary," this sentence appears: "The outstanding findings in this patient are those of pulmonary asbestosis."

## Cancer blamed

"To be sure, cancer was present, and it's not among the types of cancer which our compensation board associates with asbestos exposure."

So if you were nit-picking, legalistic, begrudging and brutish, you might argue that the cancer killed him more than the asbestosis killed him and that therefore the widow can weep empty-handed.

But in the Ontario compensation system, we have something called "benefit of the doubt" — a cardinal principle to be exercised, whenever there is uncertainty, on the worker's behalf. And surely, in the name of all that's fair, Mrs. Dodds is entitled to receive the pension which acknowledges her husband's life.

On Dec. 1, the Board sent Mrs. Dodds another letter saying that her claim had been thoroughly reviewed, and that further studies would be carried out by Dr. A. C. Ritchie (a board consultant) which "will provide us with a clearer picture of the circumstances involved in this case."

## Benefit of doubt

A clearer picture! What more do they want? If benefit of the doubt doesn't operate in this case, they should bury it with John Dodds.

When will Mrs. Dodds know? By Christmas? By 1979? Must it take so long? The board has more than 2,400 employees. Cannot one of them be found to settle this claim? The total of all benefits paid out by the board in 1977 was \$372.4 million. Mrs. Dodds would be entitled to \$365 a month.

Please, Mr. Starr, order that it be done.





# Starr says Lewis 'assumes' asbestosis caused death

□ John Dadds, 58, died a pain-wracked death after 23 years of working with asbestos. Star columnist Stephen Lewis recently blasted the Workmen's Compensation Board for its delay in compensating Dadds' widow. Its chairman, Michael Starr, in a letter that appears below, says Lewis hasn't all the facts... and Lewis answers back.

## The Editor

The Toronto Star

Dear Sir:

Stephen Lewis' failure to present an accurate report of the circumstances concerning the death of John Dadds and

the entitlements of his widow to dependency benefits (The Star, Dec. 15) leads me to believe he is seriously lacking in journalistic responsibility.

Mr. Lewis states that Mr. Dadds died as a result of exposure to asbestos at his place of work. Not once did he seek to confirm this claim with either my office or with the WCB's executive director of medical services.

Mr. Lewis assumes from his "documents" that the immediate cause of death was asbestosis. The medical information obtained by the board indicates the primary cause of death was related to a thyroid cancer, and this has been verified by contact with the pathologist who performed the autopsy. I regret very much that Mr. Lewis' article has caused a public argument to be waged regarding Mr. Dadds' illness but I feel we must refute allegations that the board is failing to respond in a humane manner to the Dadds claim.

Because Mr. Dadds' cause of death is not as black and white as Mr. Lewis states, a complete and detailed evaluation of all the facts is absolutely essential to determine compensation and possible dependency benefits for Mrs. Dadds. The board awarded Mr. Dadds a 25 per cent disability award in 1975. In May 1976, that percentage was increased to 40 per cent. At no time did Mr. Dadds ever receive a 100 per cent disability award as stated by Mr. Lewis solely on the basis of his asbestosis.

The Workmen's Compensation Act

empowers the board to grant supplementary benefits to an injured worker who is making every effort to find himself or herself suitable employment. Essentially, that entails co-operating with the worker's rehabilitation counselor.

In Mr. Dadds' case, he met our criteria and received a temporary supplementary award of 75 per cent in 1975 and a 60 per cent supplementary award following the review of May 1976, until the time of his death.

Charles Neilson, president of the union of which Mr. Dadds was a member, has been involved in this case. Unfortunately, he supplied the board

with incorrect information. He insisted that the autopsy report had been sent to the board and, still later, had been sent to the consulting pathologist expert in these matters, neither of which was factual. It was only after the medical staff at the board intervened that the copy of the autopsy report was obtained and sent to the consulting pathologist on Nov. 30. The board expects his report within a week.

An industrial disease claims specialist notified Mr. Neilson on Dec. 1 and again, on Dec. 5, as to the full status of the claim. Mr. Neilson must have either failed to understand this or ignored the information when he discussed this claim with Mr. Lewis.

Despite the overwhelming evidence which the board has, indicating the cause of death was non-compensable, we are, nevertheless, obtaining this additional information in order to provide every benefit of doubt to Mrs. Dadds' claim.

Far be it from burying the head-in-the-sand principle with Mr. Dadds' remains a vital and vibrant part of which is applied in resolving every complex industrial disease claim. This one certainly was.

Yours sincerely,  
MICHAEL STARR  
Chairman  
The Workmen's  
Compensation Board

## And Lewis quotes doctors' reports that say it WAS asbestosis

There'll be no beating around the bush in this reply to Michael Starr. If I'd known before what I know now about the John Dadds case, my original column would have been a darn sight stronger.

Mr. Starr may question my "journalistic responsibility" but one of the reasons I no longer seek confirmation from the board is exemplified, perfectly, by Mr. Starr's letter. If I were to accept, at face value, what the board has alleged over the years, I'd have spent my life down garden paths.

The crux of Mr. Starr's letter is contained in the third paragraph. Everything else can be answered. But the crucial imperative — whether or not Mrs. Dadds gets her pension — depends on whether or not the asbestosis, which John Dadds contracted after 23½ years at Johns-Manville, played a part in his death.

That's really the issue between Michael Starr and me. All else pales. If asbestosis was not at all responsible, as Mr. Starr suggests, then there's no pension. If there is good reason to believe that it was responsible, then the over-riding principle of giving the worker the "benefit of the doubt" would apply. Mrs. Dadds would get her pension. \$365 a

month, in memory of a husband who died at 58.

Mr. Starr says that I assume from my "documents" that the immediate cause of death was asbestosis. He's right.

I have in front of me a document entitled Doctor's Report of Death, filed by Dr. Christopher Pinto, under the provisions of the Workmen's Compensation Act. Beneath the heading Cause of Death, it says: "Immediate Cause — state the disease, injury or complication which caused death," and beside it — "Asbestosis."

In his letter, Mr. Starr emphasizes thyroid cancer. That is mentioned in the report as a factor "contributing to death but not... related to immediate cause."

## Autopsy report

Death certificates can be fallible. So let's turn to another document — the autopsy report. The autopsy was conducted at the Alax-Pickering hospital by Dr. Isadore Cass, a highly regarded pathologist. The four pages of findings read like some ghastly organic invasion by asbestosis. The first sentence of the summary is as follows: "The outstanding findings in this patient are those of pulmonary asbestosis."

The autopsy report also mentions thyroid cancer. That is mentioned in the report as a factor "contributing to death but not... related to immediate cause."

You can imagine my surprise, therefore, when I read Mr. Starr's letter and learned that the "primary cause" of death was related to a thyroid cancer, and was verified by the attending pathologist, in other words Dr. Isadore Cass. I simply wasn't able to reconcile the autopsy report with the Michael Starr letter.

So I took the inconsistency by the horns and phoned Dr. Cass. I think I do him complete justice in saying that Dr. Cass re-emphasized his conviction that asbestosis played the most significant part in the death, and that he had informed the board of his opinions.

Then Dr. Cass raised something of



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which I was unaware. He pointed out that in the earliest stages of thyroid cancer, surgery was one of the procedures which might be considered, but in this case, any operation would have been too great a risk because of the severe respiratory damage from asbestosis. Obviously, then, in that sense as well, asbestosis contributed to the eventual death.

Moreover, suggested Dr. Cass, if I was still perplexed, and wanted a further opinion, perhaps I should phone Dr. John S. Carruthers, the pathologist at Princess Margaret Hospital who had carefully re-examined the tissues and body sections which were sent to him after the autopsy.

I did. Just yesterday. And again I think I'm entirely fair to Dr. Carruthers when I say that he agreed, on virtually everything, with Dr. Cass.

In fact, Dr. Carruthers also drew my attention to something I had missed. He found it difficult to understand how thyroid cancer could be the primary cause of death when the radiation had already killed all the cancer cells in the neck, leaving only one fragment in the lung. Asbestosis seemed far more plausible, and it was one of the most severe asbestosis cases which Dr. Carruthers had encountered.

As if that wasn't enough, I spoke last night with Dr. Colin Syme, who had been with Dr. Carruthers attending physician for the last years of his life. He told me that the board had phoned him just last week to ask about the cause of death, and he had told them, unequivocally, that in his opinion asbestosis was overwhelmingly responsible.

We are left, then, with only the dissenting voice of the board itself, supported by its distinguished pathologist-consultant in such cases, Dr. Alex Ritchie of the University of Toronto. It is Dr. Ritchie's report for which Mr. Starr says he is waiting.

## Defence rests

I now have a copy of that report and have spoken to Dr. Ritchie. There is no question that Dr. Ritchie has very strong reservations about the impact of asbestosis on John Dadds' death. There is also no question that he feels there is other important information to be assessed — x-rays and pulmonary data, for example.

Just the same, in his report to the board, dated Dec. 18, Dr. Ritchie indicates that the presence of asbestosis in the body sections which he examined is "severe... and extensive." He did not see the whole of the lungs themselves,

and therefore cannot make further assumptions. He confirms the presence of tumor in one lung, but recognizes no living tumor in the thyroid.

But Dr. Ritchie is scrupulously scrupulous. He makes his findings without comment. He makes his findings without recommendation. The board will draw its own conclusions.

Is not this a classic case of "benefit of the doubt"?

Mr. Starr writes, in his second last paragraph, that the board has "overwhelming evidence that the cause of death was non-compensable. He's right. Finally, that asbestosis was not involved. What arrant nonsense. I could as easily write of overwhelming evidence to the contrary.

What we have here is a different opinion between the board and all the doctors, but a very strong presumption that asbestosis was a signal culprit in the death of John Dadds. Not black and white, but arguable, persuasive.

And that's what "benefit of the doubt" is all about. Where it exists, as it clearly does in this case, the board should tilt in favor of John Dadds' wife.

The defence rests, Mr. Starr. But how many other widows are waiting as the board applies its fair and impartial judgment?

